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CWK - H02358 - 9 - P024811

AJOPAA 87 (1) 1-120 (1979)

VOLUME 87. NUMBER 1 JANUARY, 1979

AMERICAN JOURNAL OF OPHTHALMOLOGY

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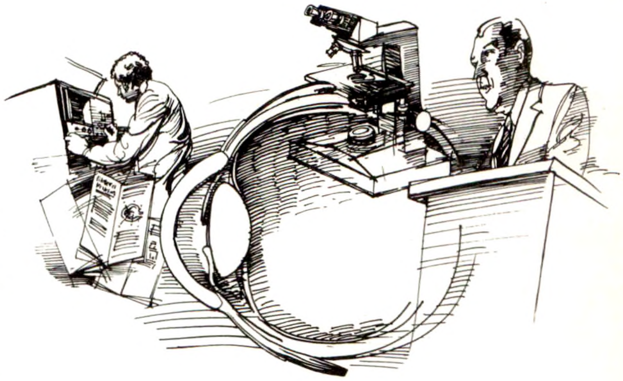

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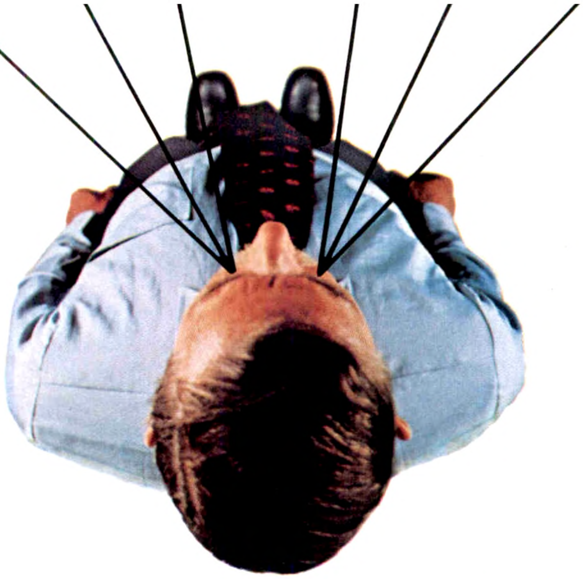

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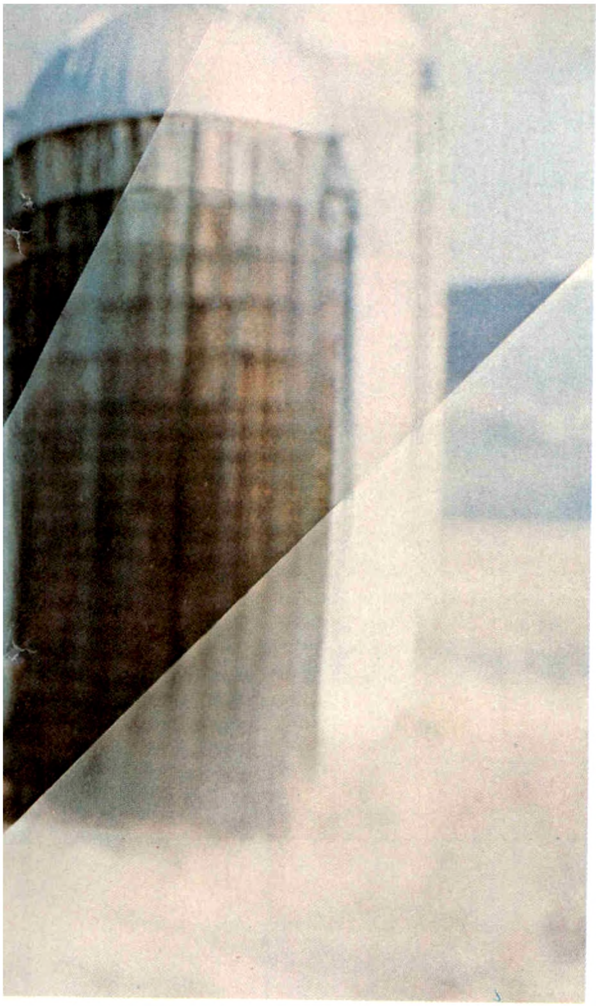

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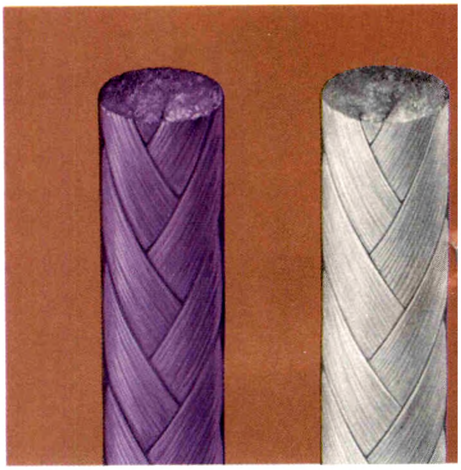

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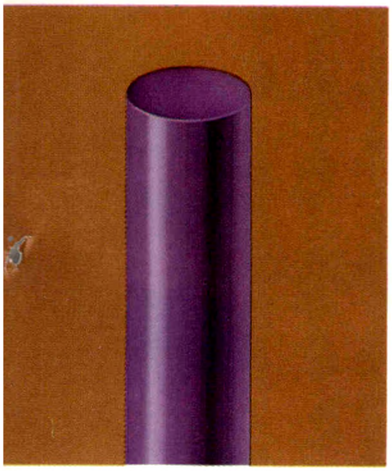

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COMPLETE PRODUCT INFORMATION

VICRYL* (Polyglactin 910)
Synthetic Absorbable Suture

DESCRIPTION VICRYL (polyglactin 910) synthetic absorbable suture is prepared from a copolymer of glycolide and lactide. These substances are derived respectively from glycolic and lactic acids. The empirical formula of the copolymer is $(C_2H_2O_2)m(C_3H_4O_2)n$.

VICRYL sutures are sterile, inert, nonantigenic, nonpyrogenic, and elicit only a mild tissue reaction during absorption. The braided and monofilament sutures are colored violet to enhance visibility in tissue. The braided suture is also available undyed (natural).

ACTIONS Two important characteristics describe the *in vivo* behavior of absorbable sutures: first, tensile strength retention, and second, the absorption rate (loss of mass).

Subcutaneous tissue implantation studies of VICRYL suture in rats show at two weeks post-implantation approximately 55% of its original tensile strength remains, while at three weeks approximately 20% of its original strength is retained.

Intramuscular implantation studies in rats show that the absorption of VICRYL suture is minimal until about the 40th post-implantation day. Absorption is essentially complete between the 60th and 90th days.

INDICATIONS VICRYL synthetic absorbable suture is intended for use as an absorbable suture or ligature.

CONTRAINDICATIONS This suture, being absorbable, should not be used where extended approximation of tissues under stress is required.

WARNINGS The safety and effectiveness of VICRYL (polyglactin 910) suture in neural tissue, and in cardiovascular surgery have not been established.

Under certain circumstances, notably orthopedic procedures, immobilization by external support may be employed at the discretion of the surgeon.

Do not resterilize.

PRECAUTIONS VICRYL suture knots must be properly placed to be secure. Place the first throw in precise position for the final knot, using a double loop; tie the second throw square, using horizontal tension; additional throws are advisable.

Skin and conjunctival sutures remaining in place longer than 7 days may cause localized irritation and should be removed as indicated.

Acceptable surgical practice must be followed with respect to drainage and closure of infected wounds.

ADVERSE REACTIONS Reactions reported in clinical trials which may have been suture related have been minimal. These include skin redness and induration, rare instances of hemorrhage, anastomotic leakage, wound separation in the eye, and abscesses.

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Chandler Dawson, MD
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Richard C. Troutman, M.D., New York, New York

Otolaryngology Speakers:

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Michael E. Glasscock, M.D., Nashville, Tennessee
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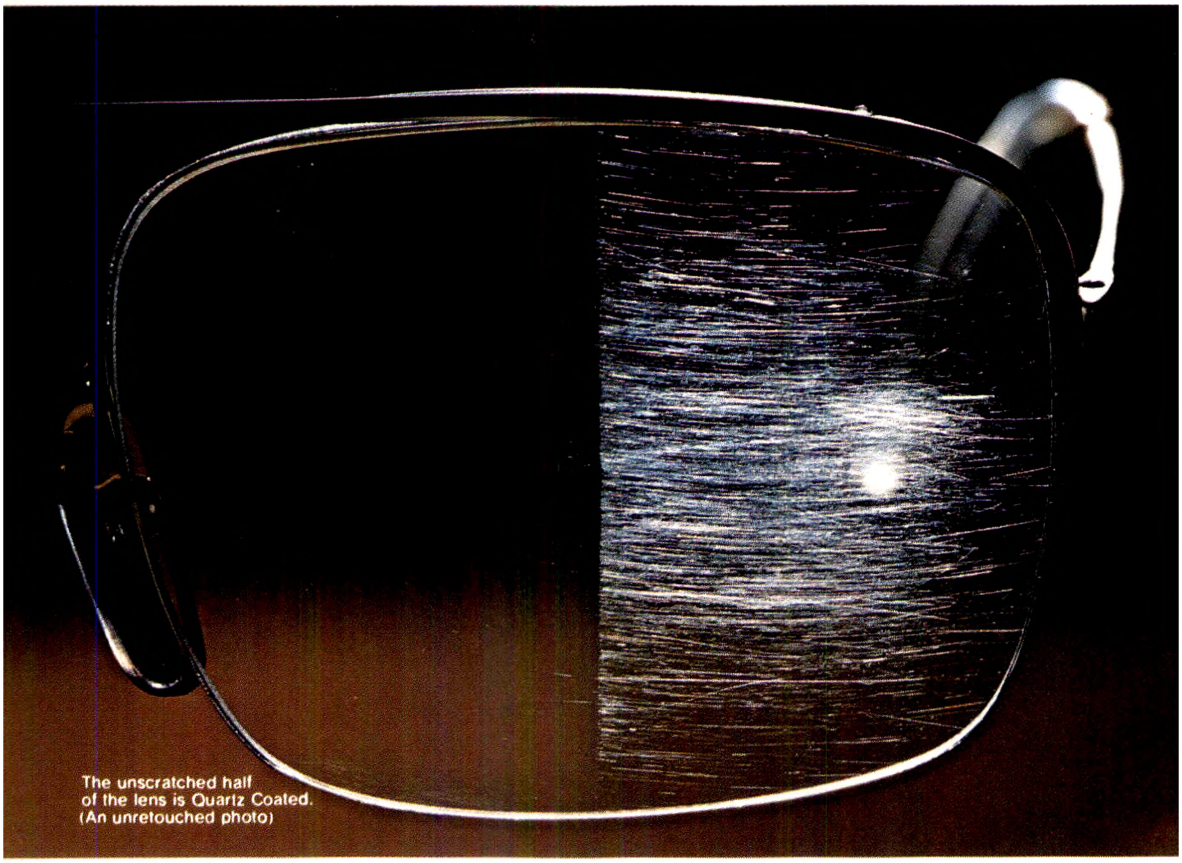
How did one side of this Hoya plastic lens get away with hardly a scratch?



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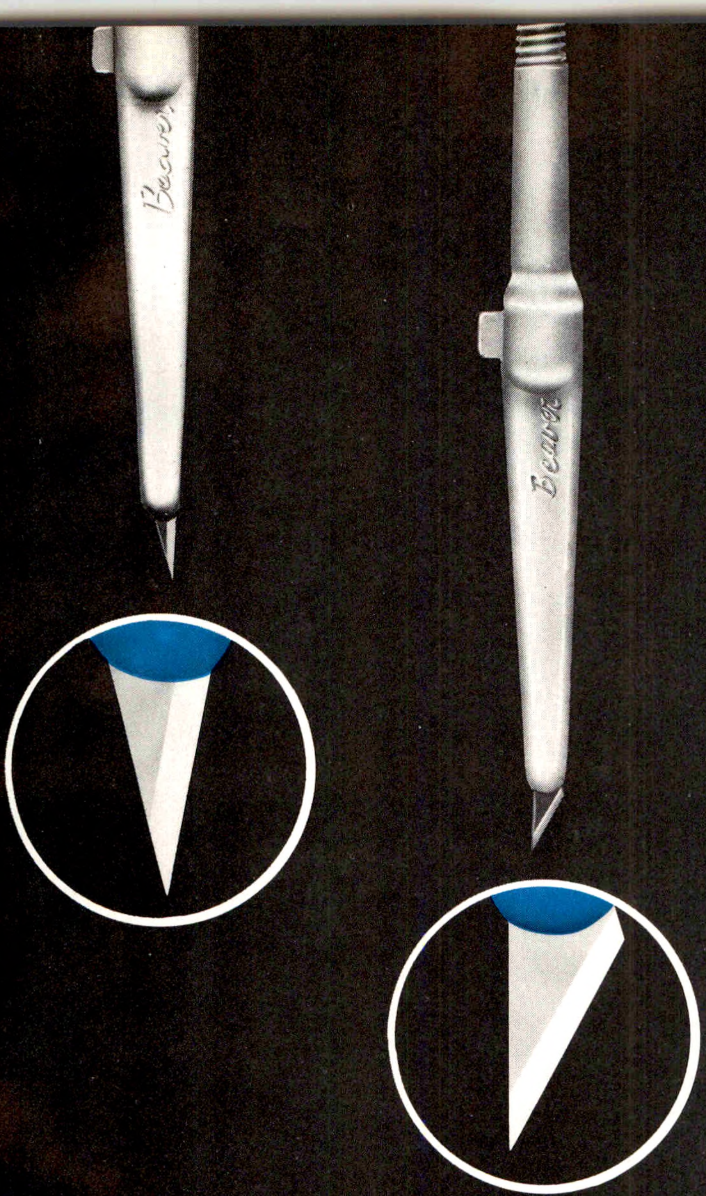

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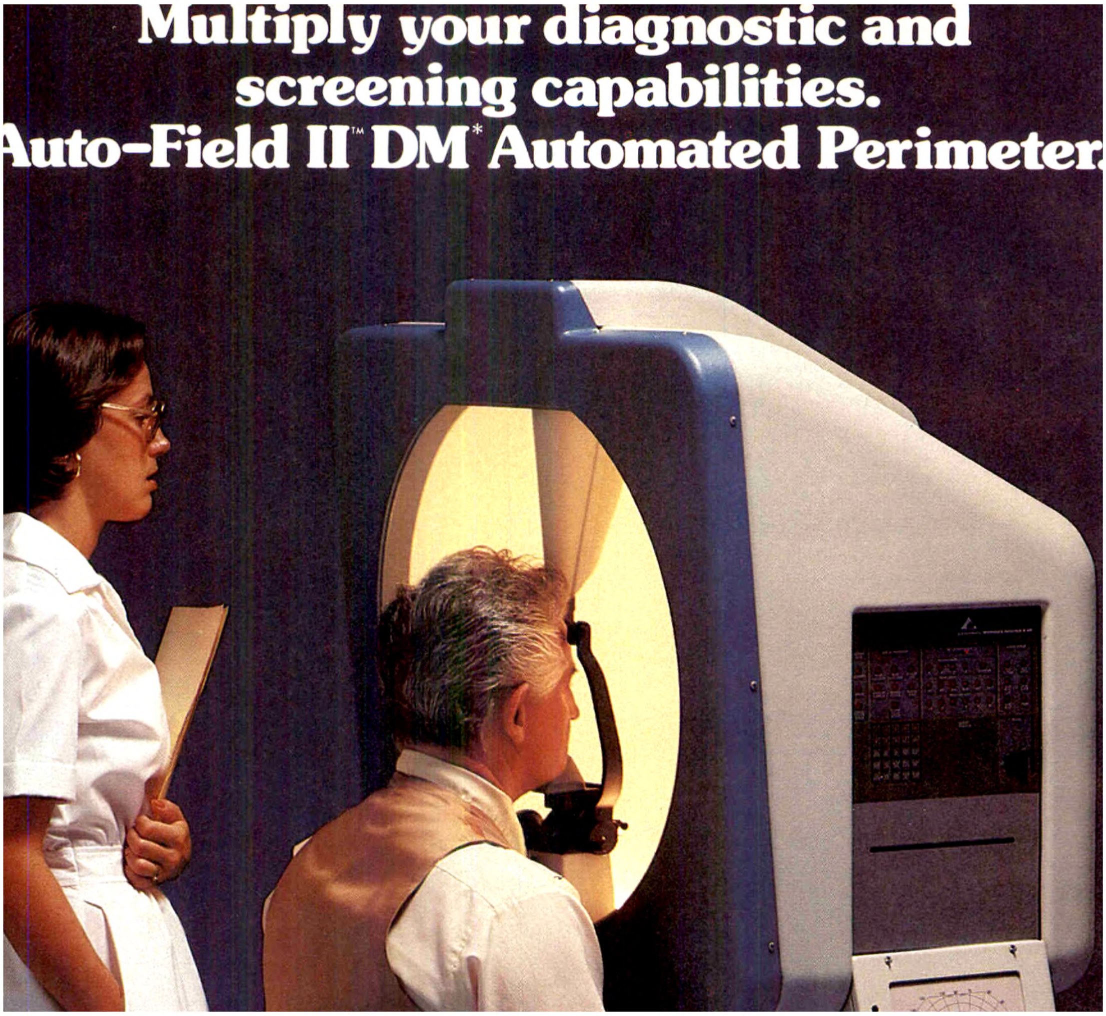

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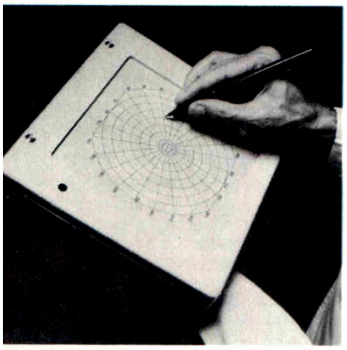

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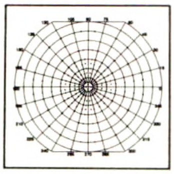

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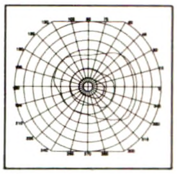

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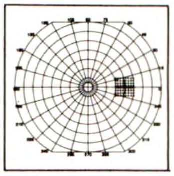

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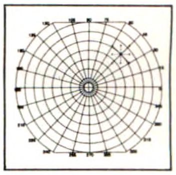

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Description. Vira-A (vidarabine, adenine arabinoside, or Ara-A) is an antiviral drug for topical ophthalmic administration. The chemical name is 9-β-D-arabinofuranosyladenine. Each gram of the ophthalmic ointment contains 30 mg of vidarabine monohydrate equivalent to 28.11 mg of vidarabine in a sterile, inert, petrolatum base.

Clinical Pharmacology. Vira-A is a purine nucleoside obtained from fermentation cultures of *Streptomyces antibioticus*. Vira-A possesses *in vitro* and *in vivo* antiviral activity against Herpes simplex types 1 and 2, Varicella-Zoster, and Vaccinia viruses. Except for Rhabdovirus and Oncornavirus, Vira-A does not display *in vitro* antiviral activity against other RNA or DNA viruses, including Adenovirus.

The antiviral mechanism of action has not been established. Vira-A appears to interfere with the early steps of viral DNA synthesis. Vira-A is rapidly deaminated to arabinosylhypoxanthine (Ara-Hx), the principal metabolite. Ara-Hx also possesses *in vitro* antiviral activity but this activity is less than that of Vira-A. Because of the low solubility of Vira-A, trace amounts of both Vira-A and Ara-Hx can be detected in the aqueous humor only if there is an epithelial defect in the cornea. If the cornea is normal, only trace amounts of Ara-Hx can be recovered from the aqueous humor.

Systemic absorption of Vira-A should not be expected to occur following ocular administration and swallowing lacrimal secretions. In laboratory animals, Vira-A is rapidly deaminated in the gastrointestinal tract to Ara-Hx.

In contrast to topical idoxuridine, Vira-A demonstrated less cellular toxicity in the regenerating corneal epithelium of the rabbit.

Indications and Usage. Vira-A Ophthalmic Ointment, 3%, is indicated for the treatment of acute keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus types 1 and 2. Vira-A is also effective in superficial keratitis caused by Herpes simplex virus which has not responded to topical idoxuridine or when toxic or hypersensitivity reactions to idoxuridine have occurred. The effectiveness of Vira-A Ophthalmic Ointment, 3%, against stromal keratitis and uveitis due to Herpes simplex virus has not been established.

The clinical diagnosis of keratitis caused by Herpes simplex virus is usually established by the presence of typical dendritic or geographic lesions on slit-lamp examination. In controlled and uncontrolled clinical trials, an average of seven and nine days of continuous Vira-A Ophthalmic Ointment, 3%, therapy was required to achieve corneal re-epithelialization. In the controlled trials, 70 of 81 subjects (86%) re-epithelialized at the end of three weeks of therapy. In the uncontrolled trials, 101 of 142 subjects (71%) re-epithelialized at the end of three weeks. Seventy-five percent of the subjects in these uncontrolled trials had either not healed previously or had developed hypersensitivity to topical idoxuridine therapy.

The following topical antibiotics: gentamicin, erythromycin, chloramphenicol; or topical steroids: prednisolone or dexamethasone, have been administered concurrently with Vira-A Ophthalmic Ointment, 3%, without an increase in adverse reactions.

Normally, corticosteroids alone are contraindicated in Herpes simplex virus infections of the eye. If Vira-A Ophthalmic Ointment, 3%, is administered concurrently with topical corticosteroid therapy, corticosteroid-induced ocular side effects must be considered. These include corticosteroid-induced glaucoma or cataract formation and progression of a bacterial or viral infection.

Vira-A is not effective against RNA virus or adenoviral ocular infections. Vira-A is also not effective against bacterial, fungal, or chlamydial infections of the cornea or nonviral trophic ulcers.

Contraindication. Vira-A Ophthalmic Ointment, 3%, is contraindicated in patients who develop hypersensitivity reactions to it.

Warnings. ***Use in Pregnancy:*** Vira-A (vidarabine) parenterally is teratogenic in rats and rabbits. Ten percent Vira-A ointment applied to 10% of the body surface during organogenesis induced fetal abnormalities in rabbits. When 10% Vira-A ointment was applied to 2 to 3% of the body surface of rabbits, no fetal abnormalities were found. This dose greatly exceeds the total recommended ophthalmic dose in humans. The possibility of embryonic or fetal damage in pregnant women receiving Vira-A (vidarabine ophthalmic ointment), 3%, is remote. The topical ophthalmic dose is small, and the drug relatively insoluble. Its ocular penetration is very low. However, a safe dose for a human embryo or fetus has not been established. Consequently, Vira-A should be used only when clearly indicated.

It is not known whether Vira-A is excreted in human milk. As a general rule, because many drugs are excreted in human milk, nursing should not be undertaken while a patient is under treatment. However, excretion of vidarabine in breast milk is unlikely because Vira-A is rapidly deaminated in the gastrointestinal tract.

Mutagenic Potential: Results of *in vitro* experiments indicate that vidarabine can be incorporated into mammalian DNA and can induce mutation in mammalian cells (mouse L5178Y cell line). Thus far, *in vivo* studies have not been as conclusive, but there is some evidence (dominant lethal assay in mice) that vidarabine may be capable of producing mutagenic effects in male germ cells.

It has also been reported that vidarabine causes chromosome breaks and gaps when added to human leukocytes *in vitro*. While the significance of these effects in terms of mutagenicity is not fully understood, there is a well-known correlation between the ability of various agents to produce such effects and their ability to produce heritable genetic damage.

Oncogenic Potential: Chronic parenteral (IM) studies of vidarabine have been conducted in mice and rats.

In the mouse study, there was a statistically significant increase in liver tumor incidence among the vidarabine-treated females. In the same study, some vidarabine-treated male mice developed kidney neoplasia. No renal tumors were found in the vehicle-treated control mice or the vidarabine-treated female mice.

In the rat study, intestinal, testicular, and thyroid neoplasia occurred with greater frequency among the vidarabine-treated animals than in the vehicle-treated controls. The increases in thyroid adenoma incidence in the high-dose (50 mg/kg) males and the low-dose (30 mg/kg) females were statistically significant.

Hepatic megalocytosis, associated with vidarabine treatment, has been found in short- and long-term rodent (rat and mouse) studies. It is not clear whether or not this represents a preneoplastic change.

The recommended frequency and duration of administration should not be exceeded (see Dosage and Administration).

Precautions. The diagnosis of keratoconjunctivitis due to Herpes simplex virus should be established clinically prior to prescribing Vira-A Ophthalmic Ointment, 3%.

Patients should be forewarned that Vira-A Ophthalmic Ointment, 3%, like any ophthalmic ointment, may produce a temporary visual haze.

Although viral resistance to Vira-A has not been observed, this possibility may exist.

Adverse Reactions. Lacrimation, foreign-body sensation, conjunctival injection, burning, irritation, superficial punctate keratitis, pain, photophobia, punctal occlusion, and sensitivity have been reported with Vira-A Ophthalmic Ointment, 3%. The following have also been reported but appear disease-related: uveitis, stromal edema, secondary glaucoma, trophic defects, corneal vascularization, and hyphema.

Overdosage. Acute massive overdosage by oral ingestion of the ophthalmic ointment has not occurred. However, the rapid deamination to arabinosylhypoxanthine should preclude any difficulty. The oral LD_{50} for vidarabine is greater than 5,020 mg/kg in mice and rats. No untoward effects should result from ingestion of the entire contents of a tube.

Overdosage by ocular instillation is unlikely because any excess should be quickly expelled from the conjunctival sac. Too frequent administration should be avoided.

Dosage and Administration. Administer approximately one half inch of Vira-A Ophthalmic Ointment, 3%, into the lower conjunctival sac five times daily at three-hour intervals.

If there are no signs of improvement after 7 days, or complete re-epithelialization has not occurred by 21 days, other forms of therapy should be considered. Some severe cases may require longer treatment.

After re-epithelialization has occurred, treatment for an additional seven days at a reduced dosage (such as twice daily) is recommended in order to prevent recurrence.

How Supplied. N 0071-3677-07 (Stock 18-1677-139)

Vira-A Ophthalmic Ointment, 3%, is supplied sterile in ophthalmic ointment tubes of 3.5 g. The base is a 60:40 mixture of solid and liquid petrolatum. **UK**

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Morris Plains, NJ 07950

PD-JA-2320-1-P (5-78)

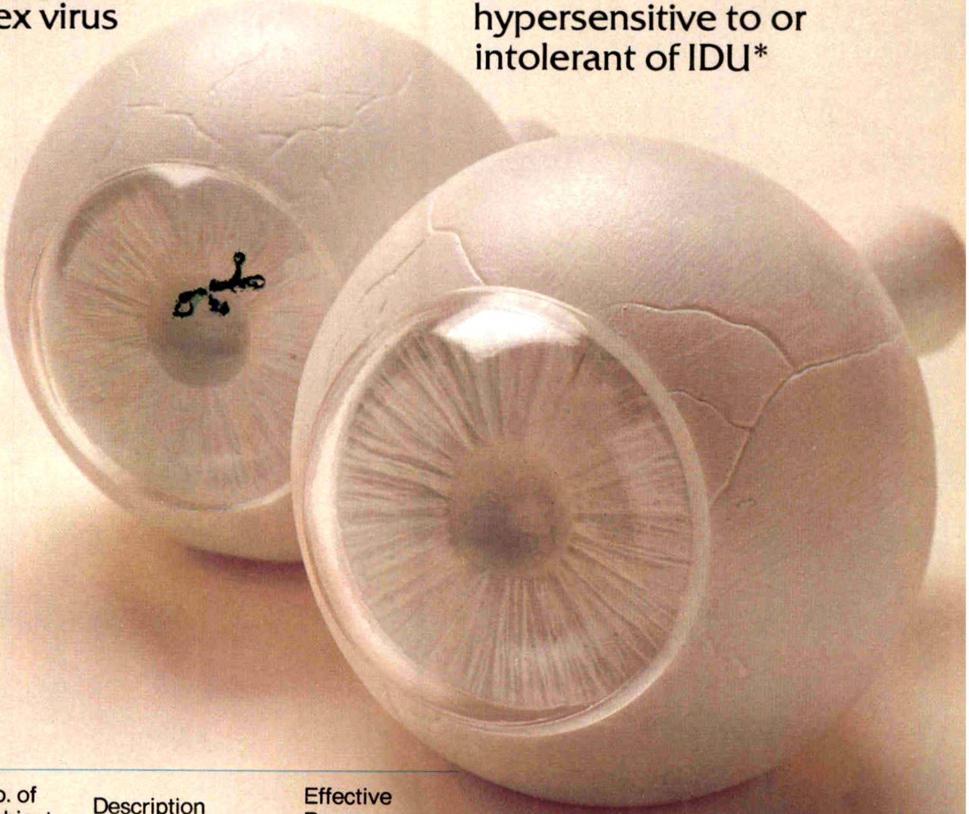
A LOGICAL FIRST CHOICE FOR HERPES SIMPLEX VIRUS KERATITIS

VIRA-A®

(vidarabine ophthalmic ointment), 3%

Not only as effective as IDU in treating Herpes simplex virus keratitis...

but is also effective in patients resistant or hypersensitive to or intolerant of IDU*



Clinical Evidence	No. of Subjects	Description	Effective Response
Vira-A Controlled Trials	81	Previously untreated	70 subjects **(86%)**
Vira-A Uncontrolled Trials	142	75% had been resistant or hypersensitive to IDU	101 subjects **(71%)**

NOTE: In the controlled and uncontrolled trials, 70 and 101 subjects, respectively, re-epithelialized at the end of three weeks on Vira-A.

Vira-A is preservative-*free*.

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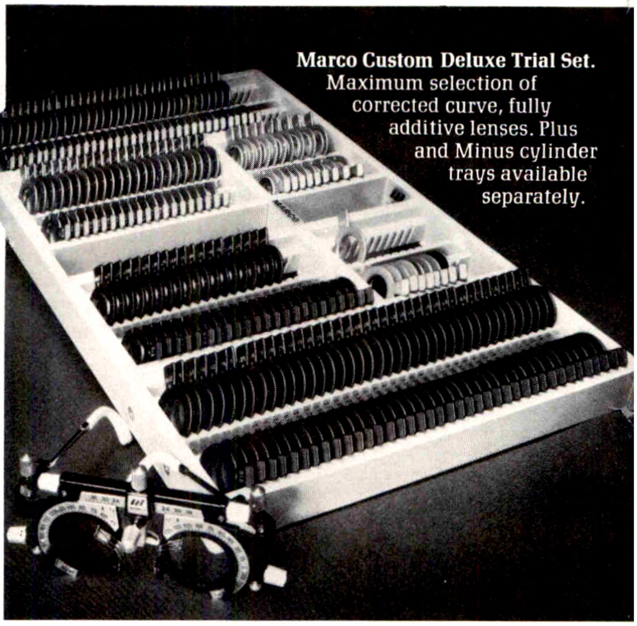
*Data on file, Medical Department, Parke-Davis.

Please see prescribing information on preceding page.

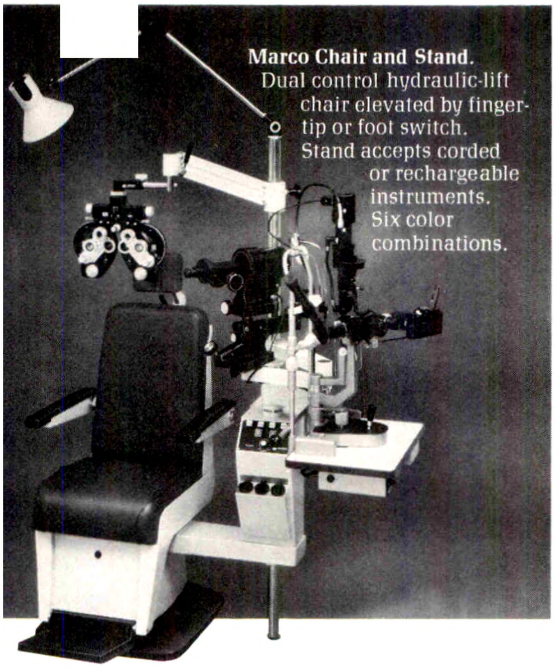
PARKE-DAVIS

Eight ways you can save

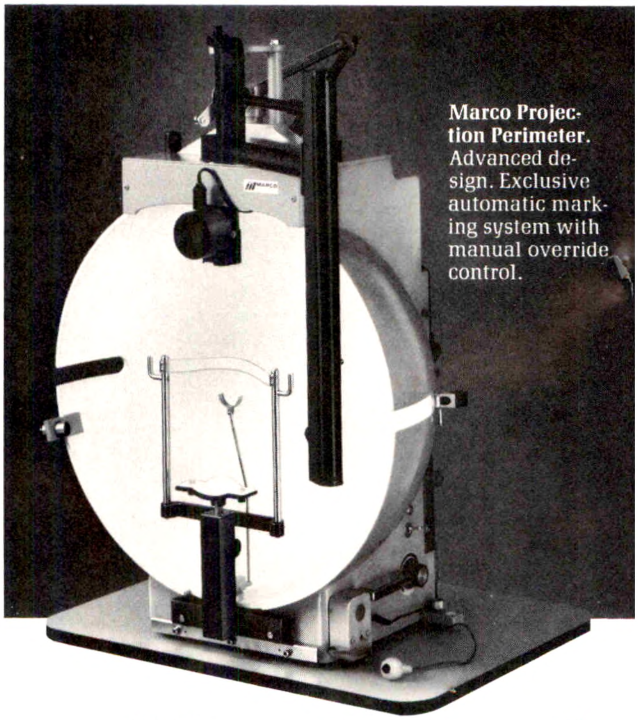
Marco Lensmeter. Full 90-degree inclination. External power and axis readings. American-style cross-line target.



Marco Custom Deluxe Trial Set. Maximum selection of corrected curve, fully additive lenses. Plus and Minus cylinder trays available separately.



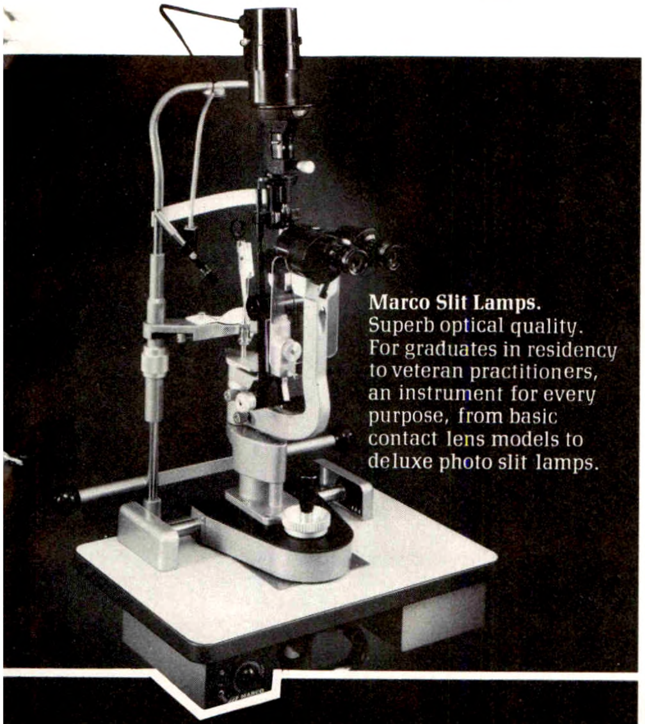
Marco Chair and Stand. Dual control hydraulic-lift chair elevated by finger-tip or foot switch. Stand accepts corded or rechargeable instruments. Six color combinations.



Marco Projection Perimeter. Advanced design. Exclusive automatic marking system with manual override control.

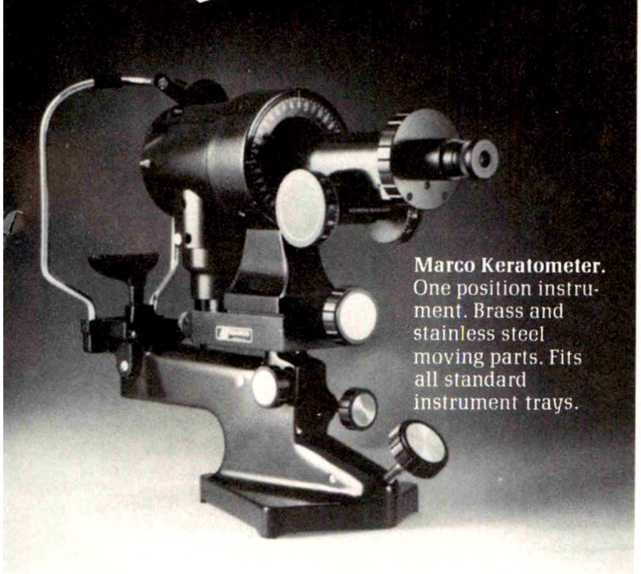
Marco puts th

when you specify Marco.



Marco Slit Lamps. Superb optical quality. For graduates in residency to veteran practitioners, an instrument for every purpose, from basic contact lens models to deluxe photo slit lamps.

Marco Chart Projector. Variable focus adjusts to any refracting distance from 10 to 20 feet. Suitable for wall, table, unit or floor mounting.



Marco Keratometer. One position instrument. Brass and stainless steel moving parts. Fits all standard instrument trays.

Marco Radiusgauge. Two instruments in one. Measures both radius and thickness with optimum accuracy through large external dial gauge and 100x magnification.

focus on value.

MARCO

Where Seeing Is Believing

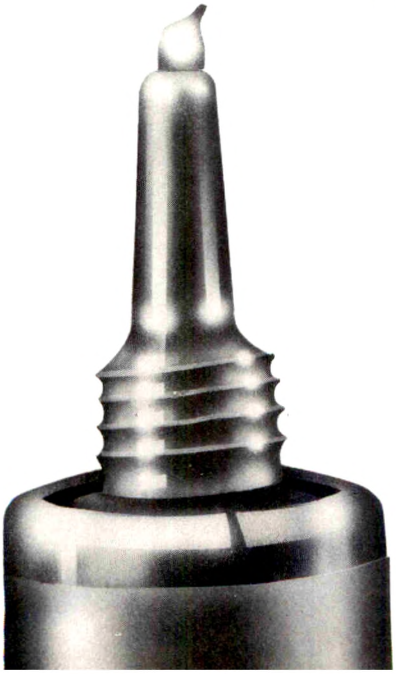
1409 San Marco Boulevard / P.O. Box 10157
Jacksonville, Florida 32207 / 904 / 396-4210

P 24,311

Cortisporin® Ophthalmic Suspension
(Sterile)

For your eyes only!

Cortisporin® Ophthalmic Ointment
(Sterile)



☐ ***Because of your specialized knowledge and training,*** we believe that only you should prescribe the Cortisporin® ophthalmic products—and that's why they are promoted only to ophthalmologists.

☐ ***For your patients*** the Cortisporin ophthalmic products provide an effective therapeutic spectrum. The antibacterials act together against susceptible bacterial pathogens commonly involved in the ophthalmic infections accessible to topical therapy.* The hydrocortisone component gently and rapidly relieves signs and symptoms of inflammation and inhibits corneal neovascularization.

☐ ***Economical, too,*** with the two sterile forms providing convenient day and night coverage at lower cost than most other major brands of similar activity.

Cortisporin® Ophthalmic Suspension Sterile

(Polymyxin B – Neomycin – Hydrocortisone)

Each cc contains Aerosporin® (Polymyxin B Sulfate) 10,000 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base), hydrocortisone 10 mg (1%), thimerosal (preservative) 0.001%. The vehicle contains the inactive ingredients cetyl alcohol, glyceryl monostearate, liquid petrolatum, polyoxyl 40 stearate, propylene glycol and purified water. Bottle of 5 cc with sterile dropper.

Cortisporin® Ophthalmic Ointment Sterile

(Polymyxin B – Bacitracin – Neomycin – Hydrocortisone)

Each gram contains Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base), hydrocortisone 10 mg (1%), special white petrolatum qs. Tube of 1/8 oz with ophthalmic tip.

***INDICATIONS:** Based on a review of these drugs by the National Academy of Sciences – National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: For the treatment of nonpurulent bacterial infections of the eye due to organisms sensitive to the antibiotic ingredients of the drugs and when the anti-inflammatory action of the hydrocortisone is indicated as in nonpurulent bacterial, allergic, vernal and phlyctenular conjunctivitis; nonpurulent blepharitis; interstitial, sclerosing, postoperative or acne rosacea keratitis; superficial chemical and thermal burns of the cornea.

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: These products are contraindicated in acute purulent conjunctivitis and blepharitis; tuberculous, fungal or viral lesions of the skin or eye, including herpes simplex, dendritic keratitis, vaccinia or varicella; and conditions involving the posterior segment of the eye. They are also contraindicated in those individuals who have shown hypersensitivity to any of their components.

PRECAUTIONS: Extended ophthalmic use of topical steroid therapy may result in glaucoma with possible damage of the optic nerves, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from fungi or viruses liberated from ocular tissues. It is advisable that intraocular pressure be checked frequently. In those diseases causing thinning of the cornea, perforation has been known to occur with the use of topical steroids. As with any antibiotic preparation, prolonged use may result in the overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Articles in the current medical literature indicate an increase in the prevalence of persons allergic to neomycin. The possibility of such a reaction should be borne in mind.

Complete literature available on request from Professional Services Dept. PML.

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

UNCOMPLICATED ORDERING.

II-2.5
NDC 0246-3010-01 CONTENTS ONE HYDRO
AOSOFT® (tetrafilco
Warning: If any eye abrasion, ulceration, irritation or infection is present, a physician should be consulted immediately.

Ordering a replacement AOSOFT® (tetrafilcon A) Hydrophilic Contact Lens is easy. Your AOSOFT distributor maintains all patient warranties, so a simple telephone call is all that's required. With the AOSOFT lens there's less paperwork for you to handle, so you have more time left to devote to your patients.

AOSOFT®
(tetrafilcon A)
HYDROPHILIC CONTACT LENSES

AO®
American Optical Corp.
SOFT CONTACT LENS DIV.
Southbridge MA 01550

Aquaflex®

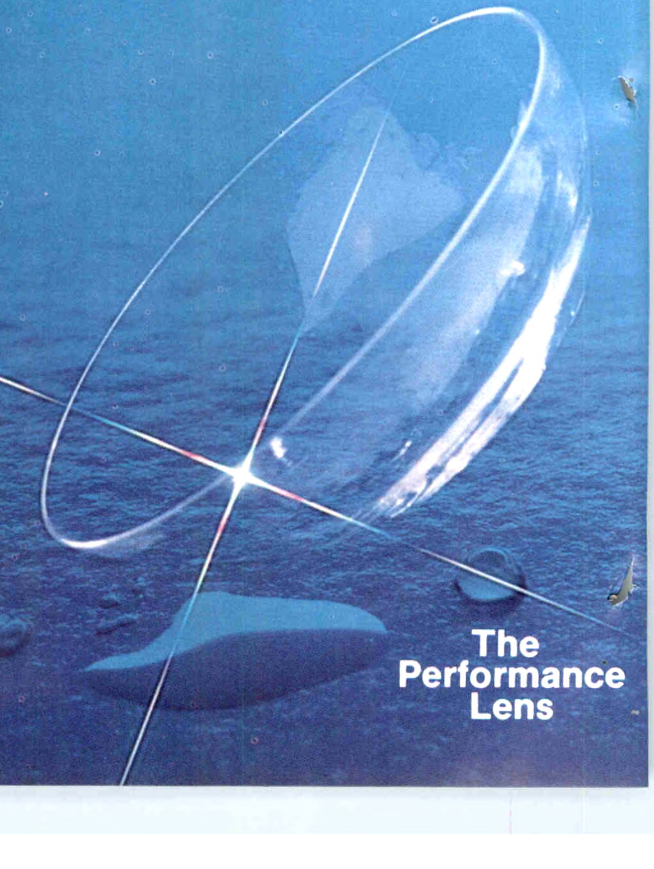
(tetrafilcon A)

Hydrophilic Contact Lens

Outstanding Visual Acuity

Optimum Centration

Predictable Fit



The Performance Story.

Soft contact lens performance that meets your exacting professional needs.

Outstanding Visual Acuity

A major benefit of Aquaflex® (tetrafilcon A) Hydrophilic Contact Lenses is excellent visual acuity. Clinical data revealed that 88% of the wearers achieved 20/20 vision or better, with 95% attaining 20/25 or better.[1] Practitioners reported that Aquaflex lenses often corrected more astigmatism and gave sharper vision than other soft lenses.[1] This quality of performance is due to a unique combination of sophisticated lens design, special lens material and the "Vault" system of fitting.

Optimum Centration

A clinical study showed that over 96% of Aquaflex contact lens wearers achieved optimum centration.[1] By centering so well, Aquaflex lenses virtually eliminate the induced astigmatism and lessened visual acuity resulting from decentered lenses.[2] Precise centration helps in attaining a good fit with full corneal coverage and maximum visual acuity, while permitting proper corneal respiration.

Predictable Fit

No complicated graphs, charts and calculations, because the Aquaflex "Vault" system reduces fitting variables to just one... the lens "Vault". Only 5 Vaults are needed to fit Aquaflex lenses;[3] and in clinical studies more than 90% of the patients were fitted with just 3 of these Vaults.[1] The bicurve, lathe-cut lens construction, with spherical front and back surfaces, promotes reliable fit independent of power; and the large posterior optical zone gives stability of vision with lens movement.

Additional Advantages

Other significant attributes of the Aquaflex® (tetrafilcon A) lens, contributing to practitioner success and patient acceptance, are: reliable over-refraction,[2,4] ease in handling, durability, excellent reproducibility[2] and exceptionally high standards of quality assurance, including 100% wet inspection. A study has shown that less than 5% of lenses shipped were returned because of questionable performance, discomfort, damage or defects.[1] The fitting procedure is simple and fast.[4] You can fit from a small diagnostic set, or use a dispensing inventory. Lenses may be ordered by a toll-free phone call, and UCO Optics' service is quick and dependable. More and more practitioners are using Aquaflex lenses every day. Shouldn't you?

Aquaflex®
(tetrafilcon A)
Hydrophilic Contact Lens

For more information and a copy of the Professional Fitting Guide, mail the coupon. Or, phone toll-free 800-828-4580 (in N.Y. State 800-462-4332).

See following page for full prescribing information.

UCO Optics, Inc. Aquaflex* Division
711 North Road, Scottsville, N.Y. 14546
I'm interested in Aquaflex® (tetrafilcon A) Hydrophilic Contact Lenses.
Send full information. ________ Have your representative call.
____
Name ________
Address ________
City ________
State ________
Zip ________
Phone ________
AJO

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1. Data on file: UCO Optics, Inc., Scottsville, N.Y. 14546
2. Morrison, Robert J.: International Contact Lens Clinic, Summer 1976
3. Gruber, Ellis & Gordon, Stanley: Contact Lens Forum, February 1977
4. Greenspoon, Morton K.: Contact Lens Forum, November 1977.

*Aquaflex is a registered trademark of UCO Optics, Inc.

Aquaflex®
(tetrafilcon A)
Hydrophilic Contact Lens

DESCRIPTION

AQUAFLEX* (tetrafilcon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The lens material, tetrafilcon A, is a hydrophilic random terpolymer of 2-hydroxyethylmethacrylate, N-vinyl-2-pyrrolidone and methylmethacrylate. The polymer is a three-dimensional network of terpolymer chains joined by divinylbenzene cross-links. It consists of 57.5% tetrafilcon A and 42.5% water by weight when fully hydrated in normal saline solution. Lenses have a nominal diameter of 13 mm.

ACTIONS

In its hydrated state the AQUAFLEX* Hydrophilic Contact Lens is soft and pliable. When dry, the lens becomes hard and brittle. These states are completely reversible and a lens which has been permitted to dry out will recover all of its hydrated properties when placed in normal saline for a period of two hours. When placed on the human cornea the hydrated lens acts as a refracting medium to compensate spherical ametropias. The material has a refractive index of 1.43 and the lens has a visible light transmittance greater than 97%.

INDICATIONS

AQUAFLEX* Hydrophilic Contact Lenses are indicated for the correction of vision in persons with non-diseased eyes who have spherical ametropias, corneal astigmatism of 2.50 diopters or less and/or refractive astigmatism of 2.00 diopters or less.

CONTRAINDICATIONS

AQUAFLEX* Hydrophilic Contact Lenses are contraindicated in the presence of any of the following conditions: (1) Acute and subacute inflammation of the anterior segment of the eye. (2) Any eye disease which affects the cornea or conjunctiva. (3) Insufficiency of lacrimal secretion. (4) Corneal hypoesthesia. (5) Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Medications and Eye Drops: AQUAFLEX* Lenses must be stored in an appropriate solution when off the eyes, the type of solution being dependent on the system used for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored *only* in BOILnSOAK* Solution (sterile buffered isotonic solution containing boric acid, sodium borate, sodium chloride 0.7%, preserved with Thimerosal (Lilly) 0.001% and edetate disodium 0.1%). When lenses are disinfected with the chemical disinfection system, they may be stored *only* in FLEXSOL* Disinfection and Storage Solution (sterile buffered isotonic solution of sodium chloride, sodium borate, boric acid, polyvinylpyrrolidone, polyoxyethylene and polyoxypropylene, preserved with Thimerosal (Lilly) 0.001%, chlorhexidine 0.005% and edetate disodium 0.1%).

No ophthalmic solutions or medications, including hard contact lens solutions, can be used by AQUAFLEX* Lens wearers prior to or while the lenses are in place on the eyes. Also, no solutions, including hard contact lens solutions, other than BOILnSOAK* Solution or FLEXSOL* Solution may be used on AQUAFLEX* Lenses when they are off of the eyes.

Since liquids and vapors may be absorbed by AQUAFLEX* Lenses, they should not be placed in the mouth for wetting, nor wiped with a cloth or tissue.

Abrasions and Infections: If a lens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present, or any abnormal eye condition is observed concurrently with lens wear, the lens should be removed immediately and a physician consulted.

Wearing Restrictions: AQUAFLEX* (tetrafilcon A) Hydrophilic Contact Lenses should not be worn while swimming, sleeping, or in the presence of irritating fumes or vapors.

Visual Blurring: If visual blurring occurs, the lens must be removed until the condition subsides.

Lens Care Regimen: Patients must adhere to the recommended daily care procedures for AQUAFLEX* Hydrophilic Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections.

PRECAUTIONS

Storage: AQUAFLEX* Lenses may be stored *only* in the appropriate storage solution: BOILnSOAK* Solution or FLEXSOL* Solution depending on disinfecting methods used. If left exposed to air, the lenses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in either BOILnSOAK* Solution or FLEXSOL* Solution until it returns to a soft, supple state.

Cleaning and Disinfecting: AQUAFLEX* Lenses *must* be both cleaned and disinfected daily. Separate procedures and products are needed to clean and to disinfect. Two methods of disinfection, thermal or chemical, have been shown to be equally effective. The choice of disinfection system should be made in consultation with your eye care practitioner.

Cleaning: Daily cleaning is necessary to remove mucus and other deposits which may have accumulated on the lens surface. Each time the lenses are removed from the wearer's eyes, both surfaces of the lenses must be cleaned using several drops of PREFLEX* Cleaning Solution (sterile buffered isotonic aqueous solution consisting of sodium phosphates, sodium chloride, tyloxapol, hydroxyethylcellulose and polyvinyl alcohol with Thimerosal (Lilly) 0.004% and edetate disodium 0.2% added as preservatives). Lenses must be cleaned before they are disinfected, as deposits on the lenses tend to harden and become more difficult to remove after the lenses are disinfected.

Disinfecting: AQUAFLEX* Lenses may be disinfected with either a heat or chemical regimen. One method or the other must be selected, but not both. The user must not alternate between methods.

Thermal Disinfection Method: AQUAFLEX* Lenses may be effectively disinfected after cleaning with PREFLEX* Cleaning Solution with use of the AQUASEPT* Patient Unit and BOILnSOAK* Solution. Fresh BOILnSOAK* Solution must be used for daily storage of lenses or each time the lens is stored. The AQUASEPT* Patient Unit requires distilled water. The AQUAFLEX* Lens Storage Containers must be emptied and filled with fresh BOILnSOAK* Solution just prior to disinfecting the lenses.

Chemical Disinfection: Disinfection with PREFLEX* Cleaning Solution, FLEXSOL* Solution and NORMOL* Rinsing Solution (sterile buffered isotonic aqueous solution of sodium chloride, sodium borate and boric acid, preserved with Thimerosal (Lilly) 0.001%, edetate disodium 0.1% and chlorhexidine 0.005%) has also been shown to be an effective disinfection system for daily care of AQUAFLEX* Lenses. AQUAFLEX* Lenses must be cleaned and rinsed daily (or after wearing) with PREFLEX* Cleaning Solution and NORMOL* Rinsing Solution. The AQUAFLEX* Lens Storage Containers must be emptied and refilled with fresh FLEXSOL* Solution each time the lens is stored. Fresh FLEXSOL* Solution must be used daily for storage and disinfection. WARNING: DO NOT MIX OR ALTERNATE THE DISINFECTION AND STORAGE SYSTEMS. FLEXSOL* SOLUTION SHOULD NOT BE USED WITH HEAT.

Hygiene: Before handling the lenses, hands must be washed, rinsed thoroughly and dried with a lint-free towel. Cosmetics, lotions, soaps, oils and hand creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Fluorescein: Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with normal saline solution and wait at least one hour before replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescein.

ADVERSE REACTIONS

Serious corneal damage may result from wearing lenses which may have soaked in hard contact lens solutions. Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritation.

Very rarely a lens may adhere to an eye as a result of a patient sleeping with the lens on, or as a result of wearing a hypotonic lens. If a lens adheres for any reason, the patient may be instructed to apply a few drops of BOILnSOAK* Solution (if using a thermal disinfection regimen) or ADAPETTES* Lubricating Solution (buffered isotonic aqueous solution containing ADSORBOBASE* [polyvinylpyrrolidone with other water soluble polymers] with Thimerosal (Lilly) 0.004% and edetate disodium 0.1% added as preservatives) (If using a chemical disinfection regimen), and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema as manifested by symptoms such as rainbows or halos around light or visual blurring may occur if lenses are worn continuously for too long a time. Removal of the lenses and a rest period of at least one hour generally relieve these symptoms. If symptoms do not subside promptly, professional consultation should be obtained.

Excessive tearing, unusual eye secretions and photophobia are not normal; if these symptoms occur, the patient should be examined to determine their cause.

DOSAGE AND ADMINISTRATION

Fitting: Conventional methods of fitting contact lenses do not apply to AQUAFLEX* (tetrafilcon A) Hydrophilic Contact Lenses. For a detailed description of the fitting technique, refer to the Professional Fitting Guide for AQUAFLEX* Hydrophilic Contact Lenses, copies of which are available from: UCO Optics, Inc., Scottsville, New York 14546.

Wearing Schedule: There may be a tendency for the patient to overwear the lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to the patient.

Day	Wear Time (*Hours*)	Rest Period (*Hours*)	Wear Time (*Hours*)
1	4	2	4
2	4	2	4
3	5	2	5
4	6	2	5
5	7	2	5
6	7	1	6
7	8	1	7
8	8	1	8
9	9	1	8
10-14	10	1	balance of waking hours†
15	all waking hours		

†lenses should never be worn 24 hours a day

Lens Care and Handling: Care must be taken on the initial visit to assure that the patient is supplied with an appropriate AQUAFLEX* Patient Care Kit and fully understands all care and handling instructions for the lenses. As with any contact lens, regular recall visits are necessary to assure patient health and compliance with instructions.

How Supplied: Each lens is supplied sterile in a glass vial containing normal saline solution. The glass vial is marked with the vault number, dioptric power, and manufacturing identification number.

The AQUAFLEX* Patient Care Kit is required for lens cleaning, disinfection and storing of the lenses. The Kit may consist of either of the following:

Thermal Disinfection Regimen

AQUAFLEX* Patient Care Kit	
AQUASEPT* Patient Unit	Catalog No. A0101
AQUAFLEX* Lens Storage Container	Catalog No. A0201
PREFLEX* Cleaning Solution	Catalog No. A1201
BOILnSOAK* Solution	Catalog No. A1301
AQUAFLEX* Patient Instruction Book	Catalog No. X0102

Chemical Disinfection Regimen

AQUAFLEX* Patient Care Kit	
AQUAFLEX* Lens Storage Container	Catalog No. A0202
PREFLEX* Cleaning Solution	Catalog No. A1201
NORMOL* Rinsing Solution	Catalog No. A1401
FLEXSOL* Disinfection and Storage Solution	Catalog No. A1501
ADAPETTES* Lubricating Solution	Catalog No. A1601
AQUAFLEX* Patient Instruction Book	Catalog No. X0102

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CAUTION: Federal law prohibits dispensing without prescription.

UCO Optics, Inc. Aquaflex Division
Scottsville, New York 14546

3/77 X0302

The Eye Computer lets kids be seen, without being heard.

Little people love the Dioptron® II Eye Computer more than anybody. Because it's fast, fun and requires no response at all. An entire examination can be completed in just half a minute. Long before your youngest patients become fidgety or cranky. And you lose your patience.

Favorite tricks like memorizing eye charts and constant blinking are no problem either. Because each examination is totally objective and each blink is automatically compensated for during the measuring cycle. Even young hyperopes are accurately refracted using the Eye Computer's special Auto-Fog™ technology.

Fact is, once a few kids have discovered how fun the Eye Computer can be, word gets around fast. And that's when you'll be hearing a lot from brothers, sisters and parents. When they ask for the Eye Computer by name.

EYE COMPUTER™

Yes, Coherent,
I want more facts on the Eye Computer.
My needs are: ☐ Immediate ☐ 3-6 months
☐ Reference
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Hospital/Clinic__________
Address__________
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Telephone__________ AJO 1/79

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3210 Porter Drive, Palo Alto, CA 94304
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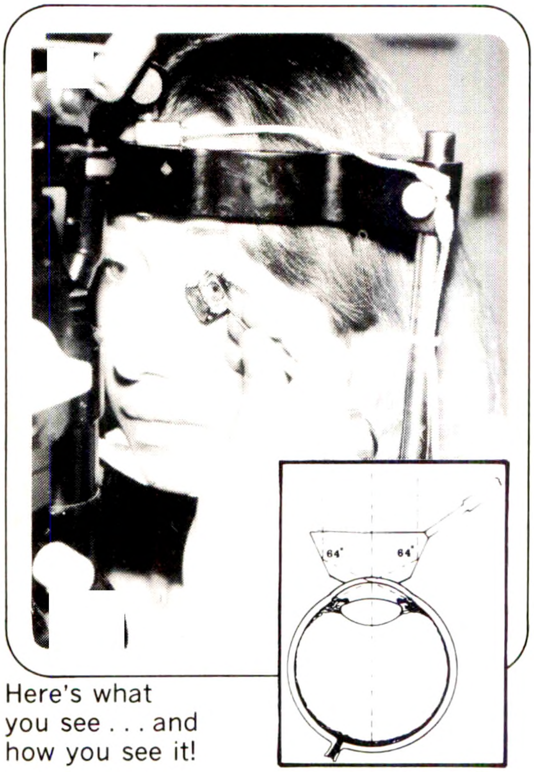
The Posner Diagnostic & Surgical Gonioprism!



Here's what
you see . . . and
how you see it!

designed by
RONALD E. POSNER, M.D. Mount Sinai Hospital

The Posner Diagnostic and Surgical Gonioprism consists of a highly-polished* silver surfaced pyramid with a plane anterior viewing surface over four mirrors, all inclined at 64°. The entire 360° angle can be observed by rotating the lens 11° in either direction while making only a slight adjustment in the slit lamp beam. It's an ideal lens for use on children or patients with a small palpabual fissure. It can also be used to evaluate surgical procedures.

Manufactured by:
Ocular Instruments, inc.
P.O. BOX 598 • REDMOND, WA. 98052 • (206) 885-1263

For more details on **AJO 1/79**
POSNER DIAGNOSTIC & SURGICAL GONIOPRISM
Write for our free catalog

NAME ________________
ADDRESS ________________
CITY ________________
STATE ________________ **ZIP** ________

Sodium SULAMYD®
brand of sodium sulfacetamide, U.S.P.
Ophthalmic Solution 30%, Solution 10%, Ointment 10% – Sterile

INDICATIONS Sodium SULAMYD is indicated for the treatment of conjunctivitis, corneal ulcer, and other superficial ocular infections due to susceptible microorganisms, and as adjunctive treatment in systemic sulfonamide therapy of trachoma.

CONTRAINDICATIONS Hypersensitivity to sulfonamide preparations contraindicates the use of Sodium SULAMYD.

PRECAUTIONS The solutions are incompatible with silver preparations. Ophthalmic ointments may retard corneal healing. Non-susceptible organisms, including fungi, may proliferate with the use of these preparations. Sulfonamides are inactivated by the para-aminobenzoic acid present in purulent exudates. Sulfonamide sensitivity reactions may occur.

June 1972 9838358

METIMYD®
brand of prednisolone acetate, U.S.P., and sulfacetamide sodium U.S.P.
Ophthalmic Suspension and Ointment – Sterile. Each ml or Gm contains 5 mg. prednisolone acetate and 100 mg. sulfacetamide sodium.

INDICATIONS – *Suspension* Based on a review of this drug by the National Academy of Sciences – National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: for the treatment of the following inflammatory and allergic conditions affecting the eyelids and anterior segment of the eye. EYELIDS: allergic blepharitis, blepharitis associated with seborrheic dermatitis and other nonpurulent forms of conjunctivitis including those associated with hay fever, and conjunctivitis due to physical agents such as foreign bodies, chemicals (acids, alkalies) and other irritants. CORNEA, SCLERA, IRIS AND UVEA: interstitial, postoperative, and sclerosing keratitis; chemical and thermal burns of the cornea; corneal ulcer; herpes zoster ophthalmicus; phylctenular keratoconjunctivitis; corneal neovascularization; scleritis; episcleritis; acute, chronic, and traumatic iridocyclitis. Final classification of the less-than-effective indications requires further investigation.

Ointment METIMYD is indicated in the following inflammatory and allergic conditions affecting the eyelids and anterior segment of the eye:
Eyelids allergic blepharitis, blepharitis associated with seborrheic dermatitis, other nonpurulent types of blepharitis.
Conjunctiva allergic conjunctivitis (ie, vernal, phylctenular) and other nonpurulent forms of conjunctivitis including those associated with hay fever, conjunctivitis due to physical agents such as foreign bodies, chemicals (acids, alkalies) and other irritants.
Cornea, Sclera, Iris, Uvea interstitial, postoperative, and sclerosing keratitis; chemical and thermal burns of the cornea; corneal ulcer; herpes zoster ophthalmicus; phlyctenular keratoconjunctivitis; corneal neovascularization; scleritis; episcleritis; acute, chronic and traumatic iridocyclitis.
In deep-seated infections, such as endophthalmitis, panophthalmitis, and orbital cellulitis, or when systemic infection threatens, specific oral (antibiotic, sulfonamide) therapy should be employed. Local treatment may be used as adjunctive therapy.

CONTRAINDICATIONS The contraindications for METIMYD are the same as those for other corticosteroid-sulfonamide preparations. Topical ophthalmic corticosteroid preparations and combinations are contraindicated in: early acute herpes simplex and the early acute stages of most other viral diseases of the cornea and conjuctiva; active tuberculosis of the anterior segment of the eye; fungal disease of the cornea, conjunctiva and lids; acute purulent untreated infections of the eye which, like other diseases caused by microorganisms, may be masked or enhanced by the presence of the steroid; individuals with known sensitivity to any of the ingredients.

PRECAUTIONS Extended use of topical steroid therapy may cause increased intraocular pressure in certain individuals. In prolonged therapy, it is advisable that intraocular pressure be checked frequently. In those diseases causing thinning of the cornea, perforation has been known to have occurred with the use of topical steroids. As with any antibacterial preparation, prolonged use may result in overgrowth of non-susceptible organisms, including fungi. If superinfection occurs, appropriate measures should be instituted. Sensitivity reactions may occur in certain individuals. If signs of sensitivity develop, discontinue use.
The protracted use of topical corticosteroids in the eye reportedly has been rarely associated with the development of posterior subcapsular cataracts.

January 1973, July 1973 9835356, 10106524

Schering

Schering Corporation
Kenilworth, N.J. 07033

SWW-9570

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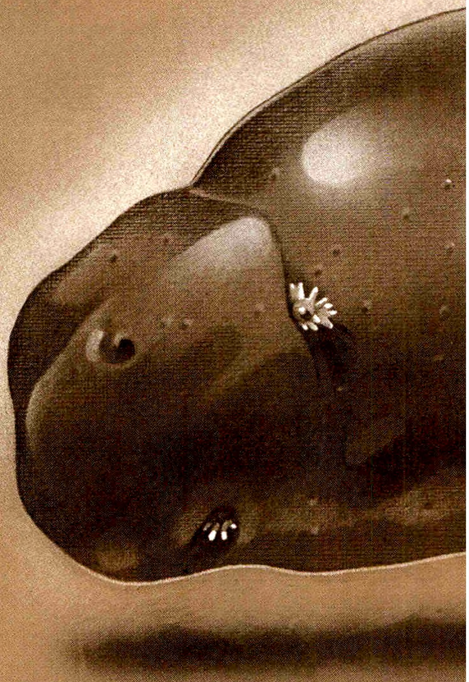

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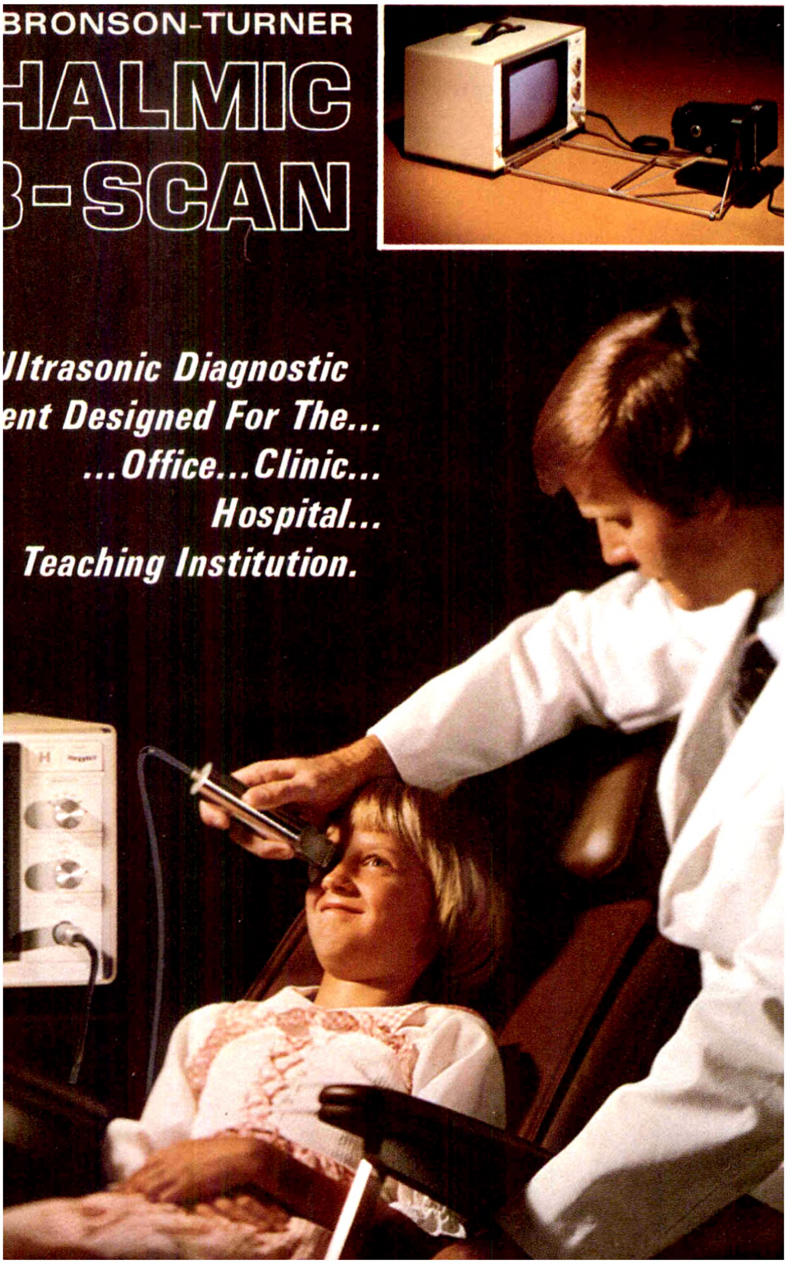

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*NDA Clinical #10-878 (1955-'57). Additional NDA Clinical #10-878 (1961). Subsequent Double-Blind Efficacy Studies (8/'72, 9/'76, & 2/'77).

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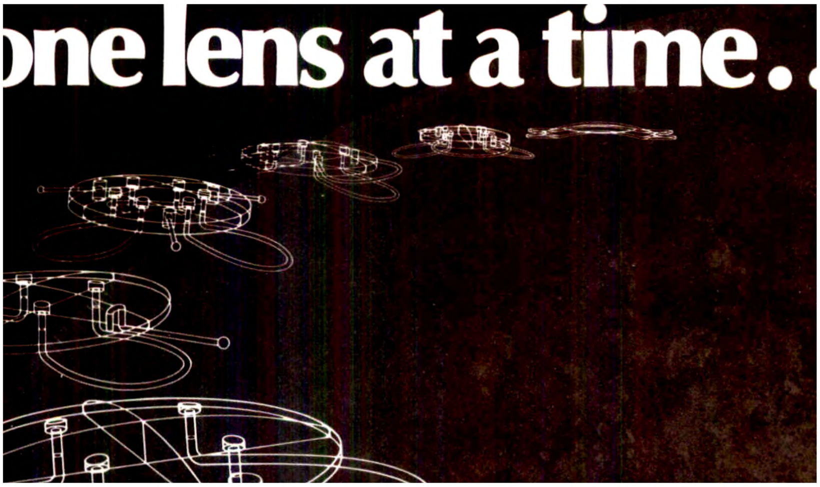
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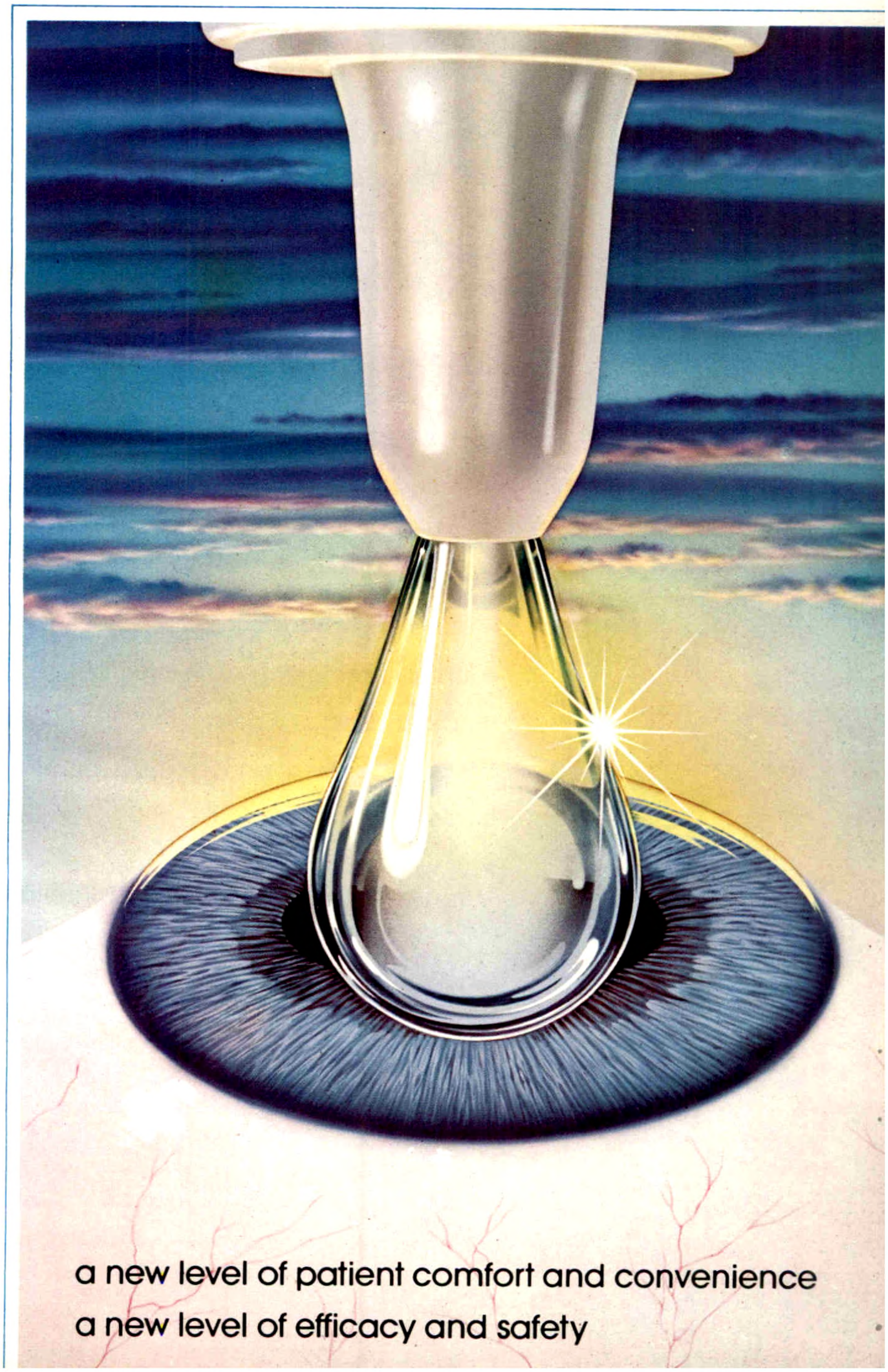
a significant advance in the topical treatment of chronic open-angle glaucoma...


a new level of patient comfort and convenience
a new level of efficacy and safety

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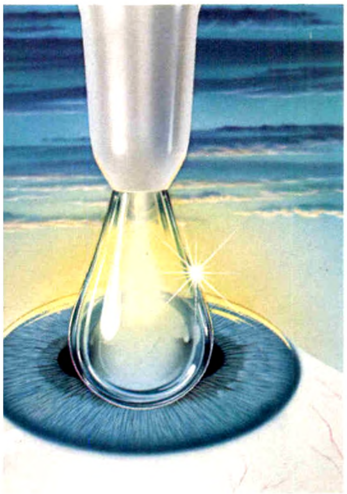


new

Timoptic™

(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION

a beta-adrenergic blocking agent in ophthalmic solution...for reduction of elevated IOP in chronic open-angle glaucoma

significantly more effective than pilocarpine or epinephrine with fewer and less severe side effects*

- little or no change in pupil size
- little or no blurring of vision
- no dimming of vision...no night blindness
- little or no effect on tear production
- little or no hyperemia
- simple "one-drop" b.i.d. dosage (may encourage long-term compliance)

TIMOPTIC™ (Timolol Maleate, MSD) is contraindicated in patients who are hypersensitive to any component of this product.

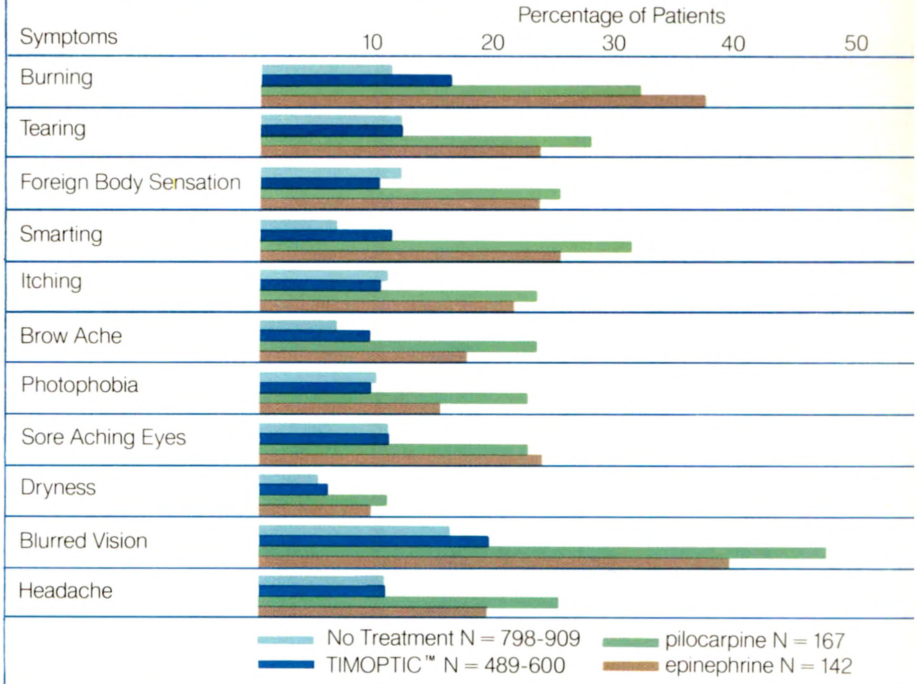
Occasionally, mild ocular irritation has been reported.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.

*Based on controlled multiclinic studies, evaluating over 1,000 patients. Data on file, Merck Sharp & Dohme. For published articles from the clinical studies on TIMOPTIC,™ please see last page of this advertisement.

a new level of patient comfort and convenience

In clinical studies—a much lower incidence of ocular discomfort than with pilocarpine or epinephrine



Adverse effects commonly caused by pilocarpine and epinephrine as reported in the literature—rarely encountered with TIMOPTIC™ (Timolol Maleate, MSD)

Drug-Induced Adverse Effects

pilocarpine	**TIMOPTIC™ (in clinical studies)**	**epinephrine**	**TIMOPTIC™ (in clinical studies)**
extreme miosis	none reported	conjunctival irritation	rare
spasm of accommodation	none reported	hyperemia	rare and mild
shallowing of the anterior chamber	none reported	macular edema in aphakic patients	none reported
night blindness and dimming of vision	none reported	pigment deposits on lid margins and conjunctiva	none reported
ciliary and conjunctival congestion	rare and mild		

With TIMOPTIC,™ slight reduction of the resting heart rate has been observed in some patients. Local hypersensitivity reactions have occurred rarely.

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new Timoptic™

(TIMOLOL MALEATE | MSD) OPHTHALMIC SOLUTION

a beta-adrenergic blocking agent
in ophthalmic solution...for reduction of
elevated IOP in chronic open-angle glaucoma

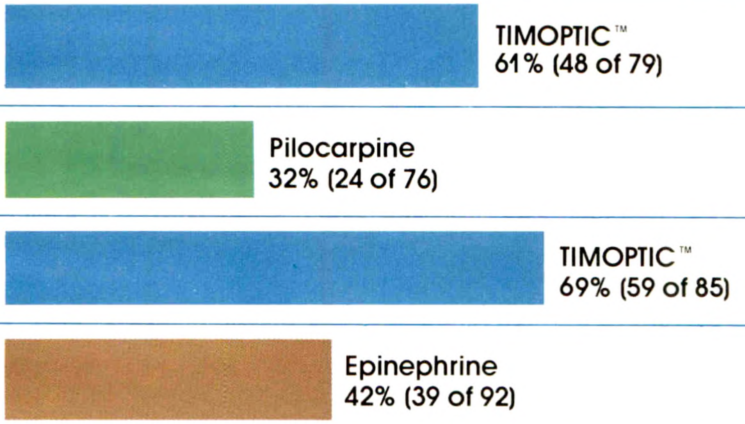
- Fast onset of action (usually within ½ hour)
- Based on clinical studies of 638 patients maintained on therapy from 14 to 477 days:
  approximately 76% (485 of 638) of patients satisfactorily managed (IOP less than 22 mm Hg) on TIMOPTIC™ alone
  approximately 93% (593 of 638) of patients satisfactorily managed with TIMOPTIC™ alone or administered concomitantly with other antiglaucoma agents
- No evidence of diminished responsiveness reported in 102 patients treated for at least two years
- Absence of miosis may facilitate visualization of optic disc and evaluation of visual fields
- Usually well tolerated
- Produced fewer and less severe side effects than either pilocarpine or epinephrine

TIMOPTIC™ should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. Patients who are already receiving a beta-adrenergic blocking agent orally should be observed for a potential additive effect.

a new level of efficacy and safety

In comparative clinical studies—significantly more effective than pilocarpine or epinephrine

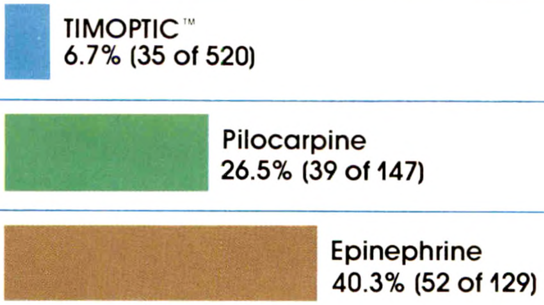
In multiclinic studies, TIMOPTIC™ (Timolol Maleate, MSD) reduced IOP in proportionately more patients



% of patients with IOP reduced to less than 22 mm Hg ($p < 0.01$)

Data are derived from the final visit (week 17) in the multiclinic pilocarpine and epinephrine comparative studies. These include patients who withdrew due to ineffective response or an adverse clinical experience.

In all clinical studies, therapy with TIMOPTIC™ resulted in fewer dropouts* than with pilocarpine or epinephrine



Data include patients participating in clinical studies lasting 14 to 477 days.

*Dropouts are defined as patients whose IOP could not be reduced to levels below 22 mm Hg, or those who withdrew because of an adverse clinical experience.

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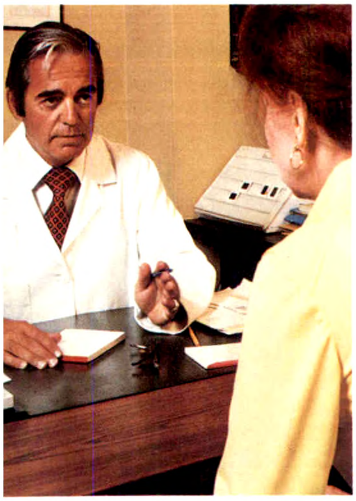
new Timoptic™

(TIMOLOL MALEATE | MSD) OPHTHALMIC SOLUTION

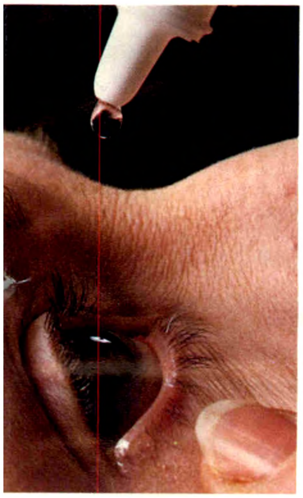


significantly broadens the medical options in antiglaucoma therapy

- **Permits prolonged maintenance therapy— no evidence of tolerance reported** In a clinical study of 102 patients treated for at least two years, there was no evidence of diminished responsiveness to TIMOPTIC™.
- **Compatible with other agents** Concomitant therapy with pilocarpine and other miotics, and/or epinephrine, and/or systemically administered carbonic anhydrase inhibitors, such as acetazolamide, can be instituted for the patient who needs reduction of IOP greater than provided by one drop of 0.5% TIMOPTIC™ twice a day. Dosages above one drop of 0.5% TIMOPTIC™ twice a day generally have not been shown to produce further reduction in intraocular pressure.
- **Helpful in the medical management of a broader range of patients** In some patients who respond inadequately to maximum available drug therapy, the addition of TIMOPTIC™ may result in satisfactory management of intraocular pressure, thus possibly postponing the need for surgery.
- **Often effective in patients previously difficult to manage**...such as working patients incapacitated by accommodative spasm produced by miotics, older patients with cataracts whose vision is impaired with miotics, patients with heavily pigmented irides whose response to pilocarpine is diminished, and aphakic patients who may develop macular edema from epinephrine.

Since clinical studies in children have not been done, TIMOPTIC™ is not recommended for use in children at this time. TIMOPTIC™ has not been studied in human pregnancy. The use of TIMOPTIC™ in pregnant women requires that the anticipated benefits be weighed against the possible hazards.

a beta-adrenergic blocking agent in ophthalmic solution...for reduction of elevated IOP in chronic open-angle glaucoma



Dosage Guidelines:

How to transfer from current therapy to TIMOPTIC™ (Timolol Maleate, MSD):

1 On the first day: continue with the agent(s) already being used and add one drop 0.25% TIMOPTIC™ in each eye b.i.d.

2 On the second day: discontinue the previously used agent(s) and continue with TIMOPTIC™ in each eye b.i.d.

How to add TIMOPTIC™ to other agents:

1 Administer one drop 0.25% TIMOPTIC™ in each eye b.i.d....while continuing with the agent(s) already being used.

2 If a higher dosage of TIMOPTIC™ is required, substitute one drop 0.5% TIMOPTIC™ in each eye b.i.d....while continuing with the agent(s) already being used.

How to start patients on TIMOPTIC™:

Usual starting dosage:
one drop 0.25% TIMOPTIC™
in each eye twice a day.

If clinical response is not adequate:

Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC™ twice a day in each eye. If the intraocular pressure is maintained at satisfactory levels, the dosage schedules may be changed to one drop once a day in each eye. Dosages above one drop of 0.5% TIMOPTIC™ twice a day generally have not been shown to produce further reduction in IOP.

℞
Timoptic™
(Timolol
Maleate | MSD)
0.25%
one drop
in each eye
b.i.d.

Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.

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• For a brief summary of Prescribing Information, please see following page.

for many patients—a significant
advance for the reduction of elevated IOP

- in chronic open-angle glaucoma
- in ocular hypertension
- in aphakic glaucoma
- in some patients with secondary glaucoma

new
Timoptic™
(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION

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11. Zimmerman, T. and Kaufman, H.: Timolol: Dose response and duration of action, Arch. Ophthalmol. *95(4)*:605-607, April 1977.

CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.

PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchial asthma; sinus bradycardia and greater than first-degree block; cardiogenic shock; right ventricular failure secondary to pulmonary hypertension; congestive heart failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.

Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.

Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.

ADVERSE REACTIONS: Occasionally, signs and symptoms of mild ocular irritation have been reported. Slight reduction of the resting heart rate (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed in some patients. Local hypersensitivity reactions have occurred rarely.

Note: TIMOPTIC has been used in patients with glaucoma wearing conventional (PMMA) hard contact lenses, and has generally been well tolerated. TIMOPTIC has not been studied in patients wearing lenses made with materials other than PMMA.

HOW SUPPLIED:

TIMOPTIC Ophthalmic Solution, 0.25%
TIMOPTIC Ophthalmic Solution, 0.5%

Both are available in 5-ml plastic OCUMETER® Ophthalmic Dispenser with a controlled drop tip.

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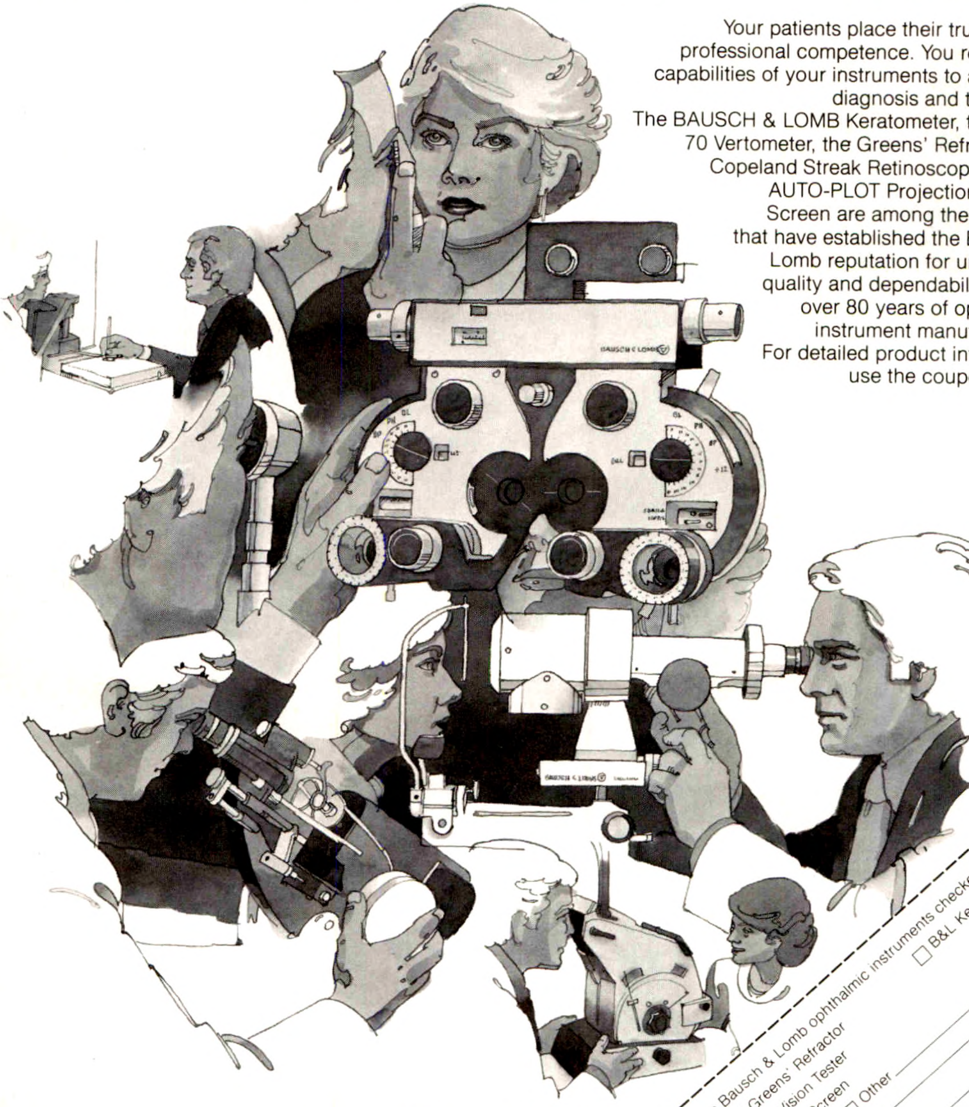

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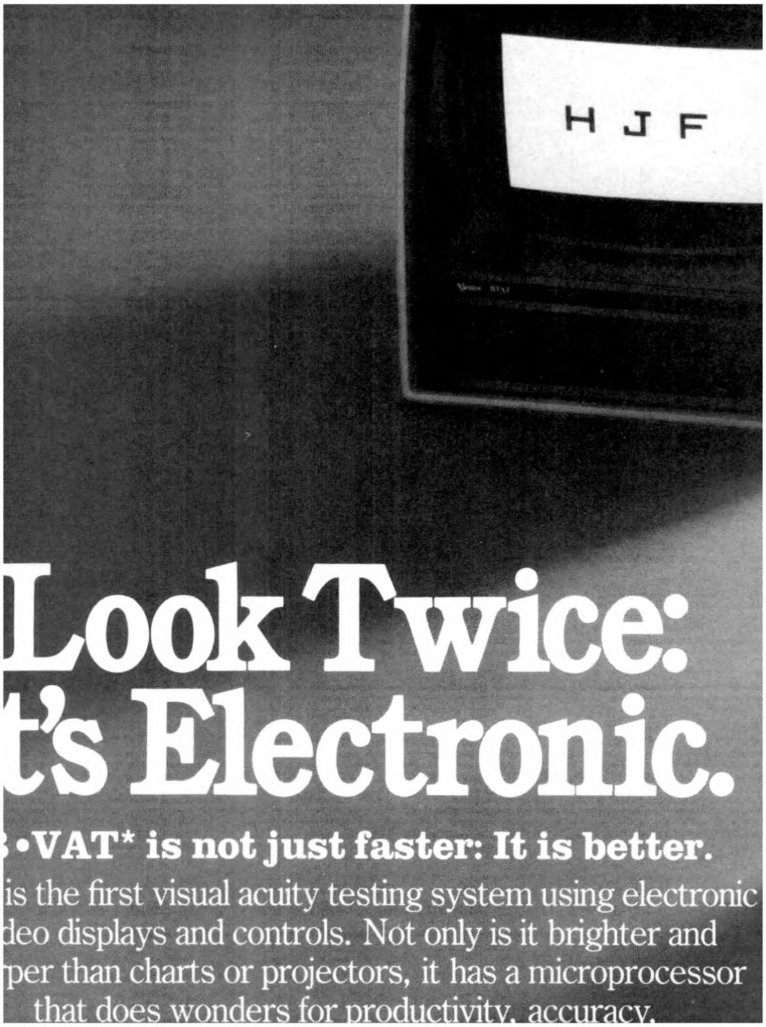

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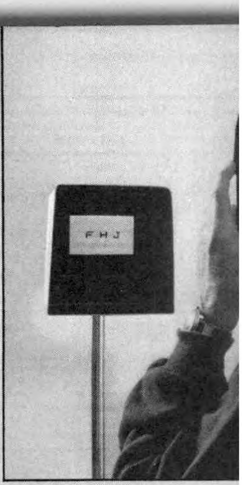

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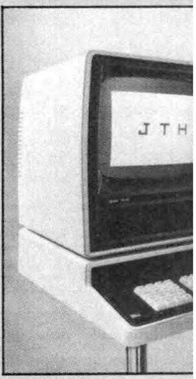

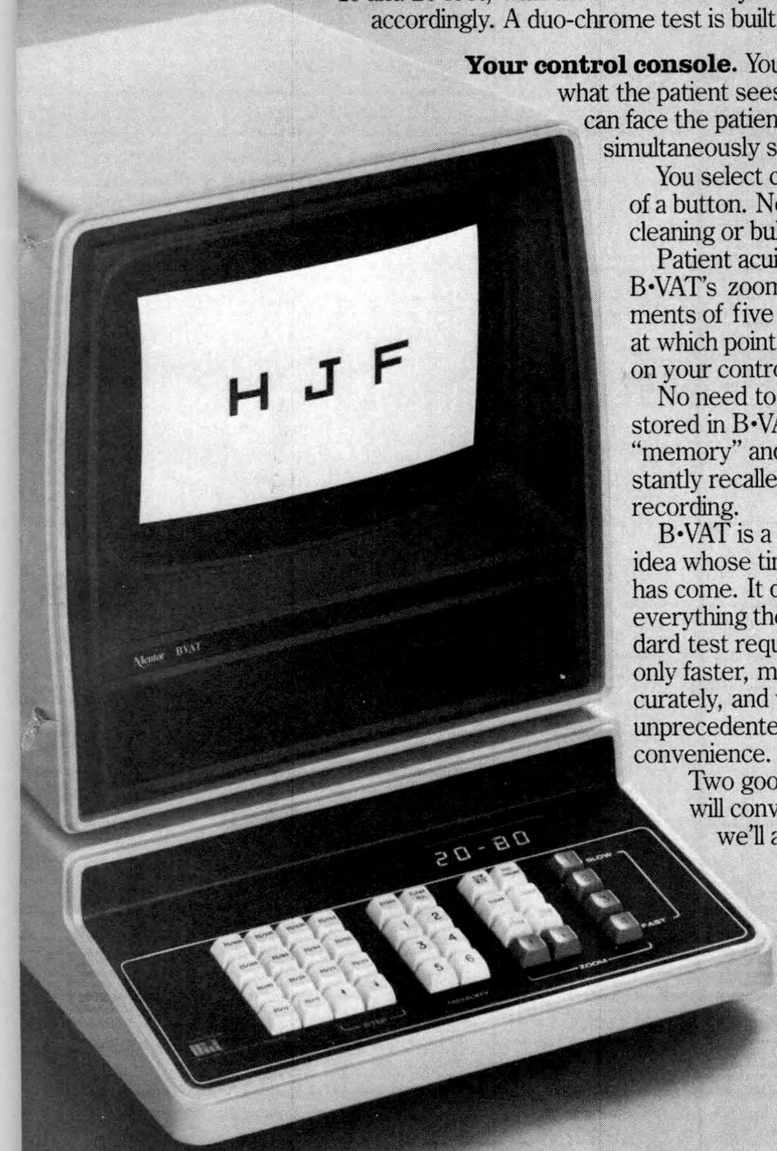

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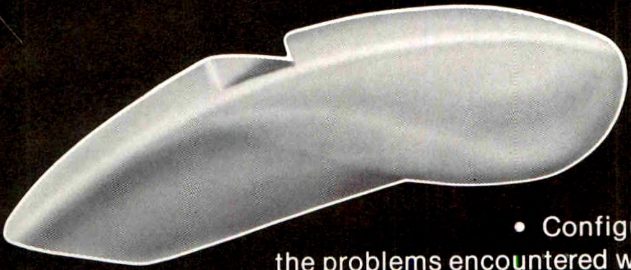

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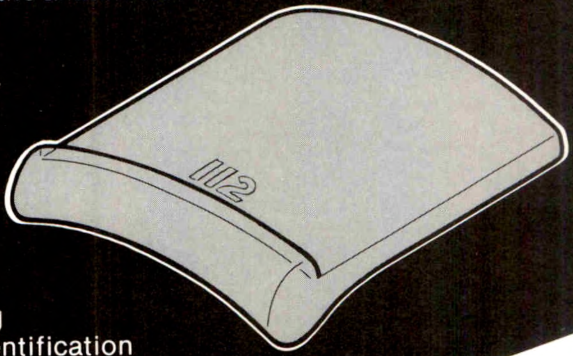

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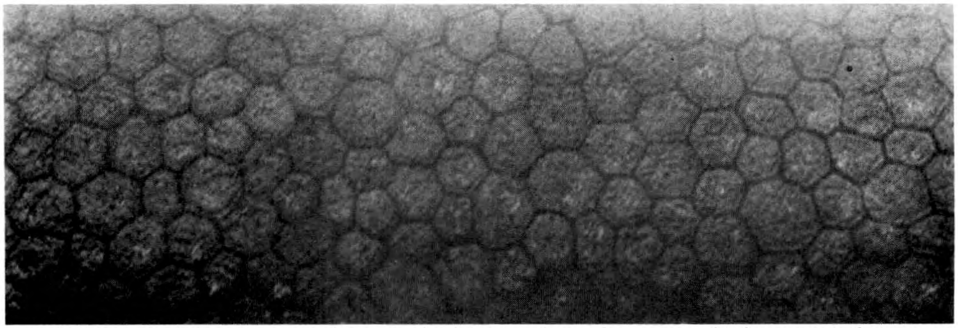

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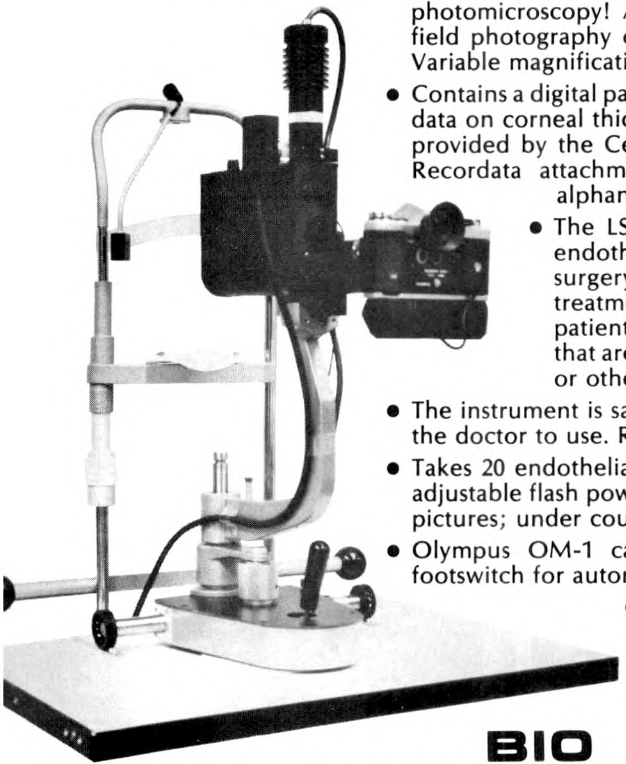

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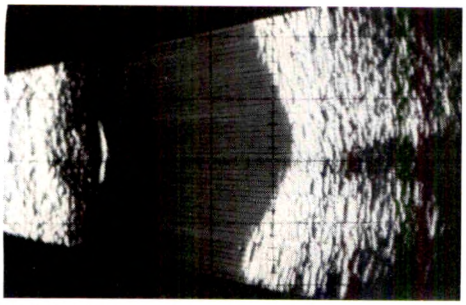
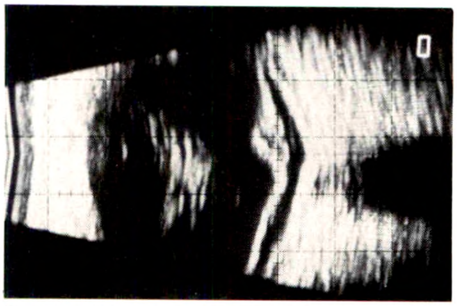


A. Contact B-scan through a closed lid showing normal posterior lens capsule and the optic nerve.



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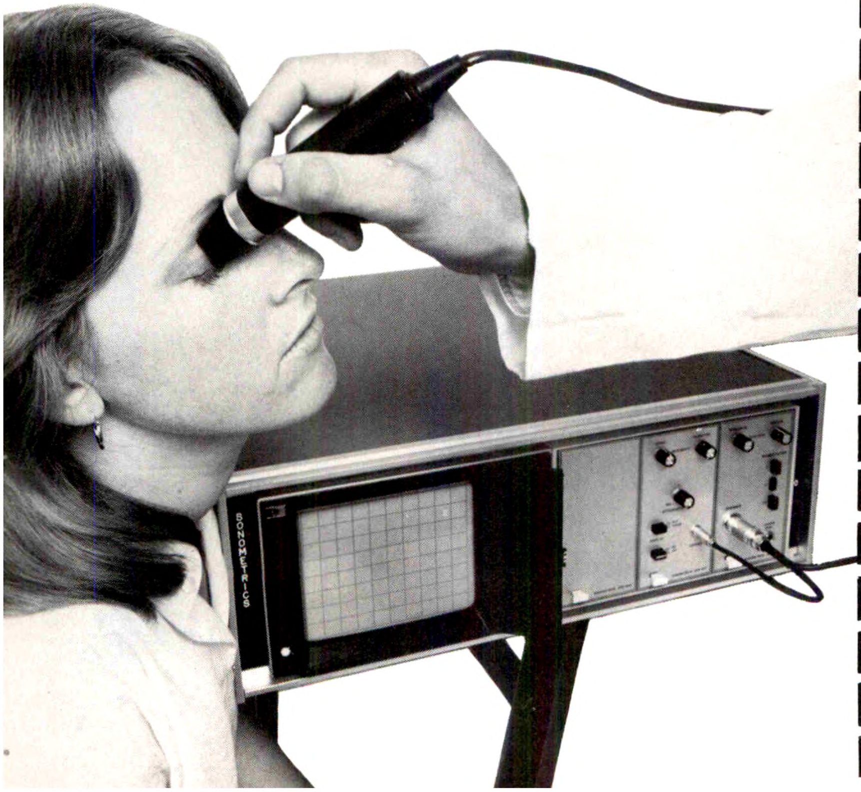

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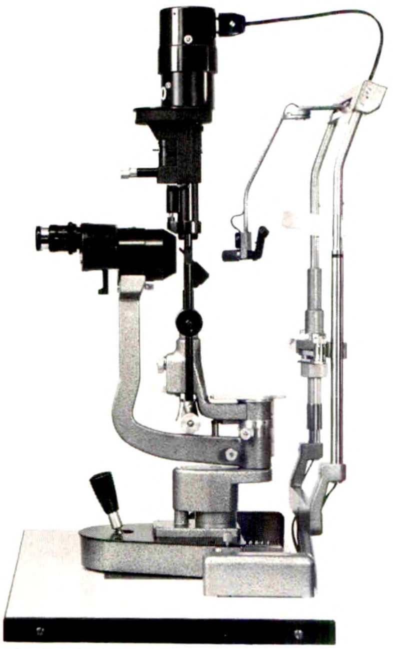

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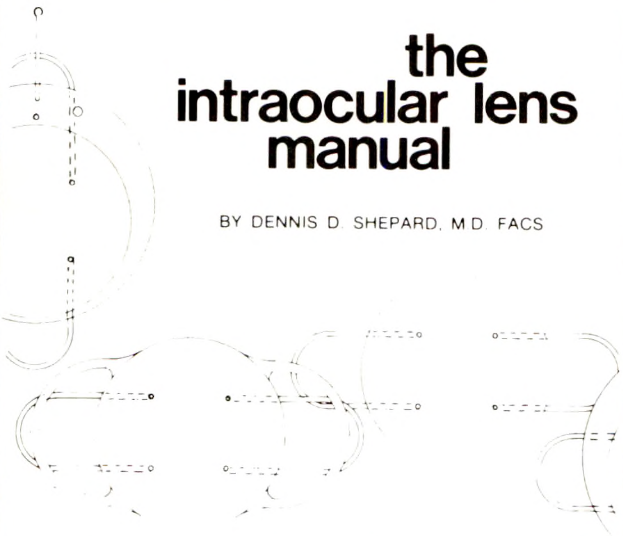

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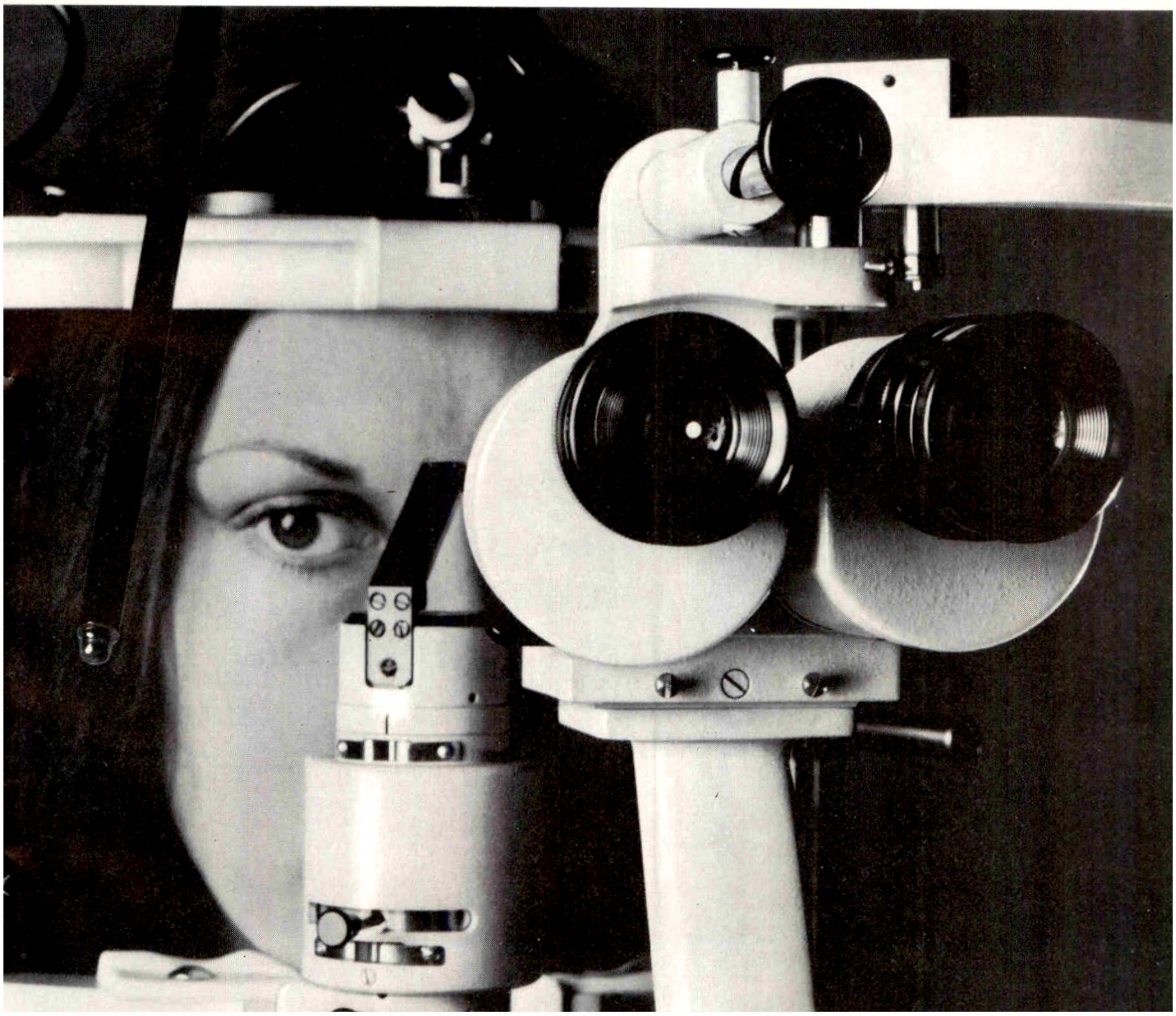

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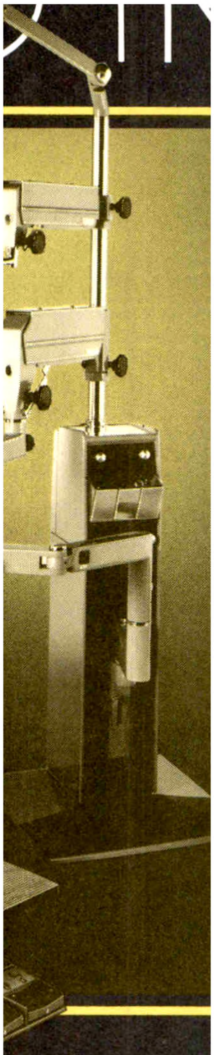

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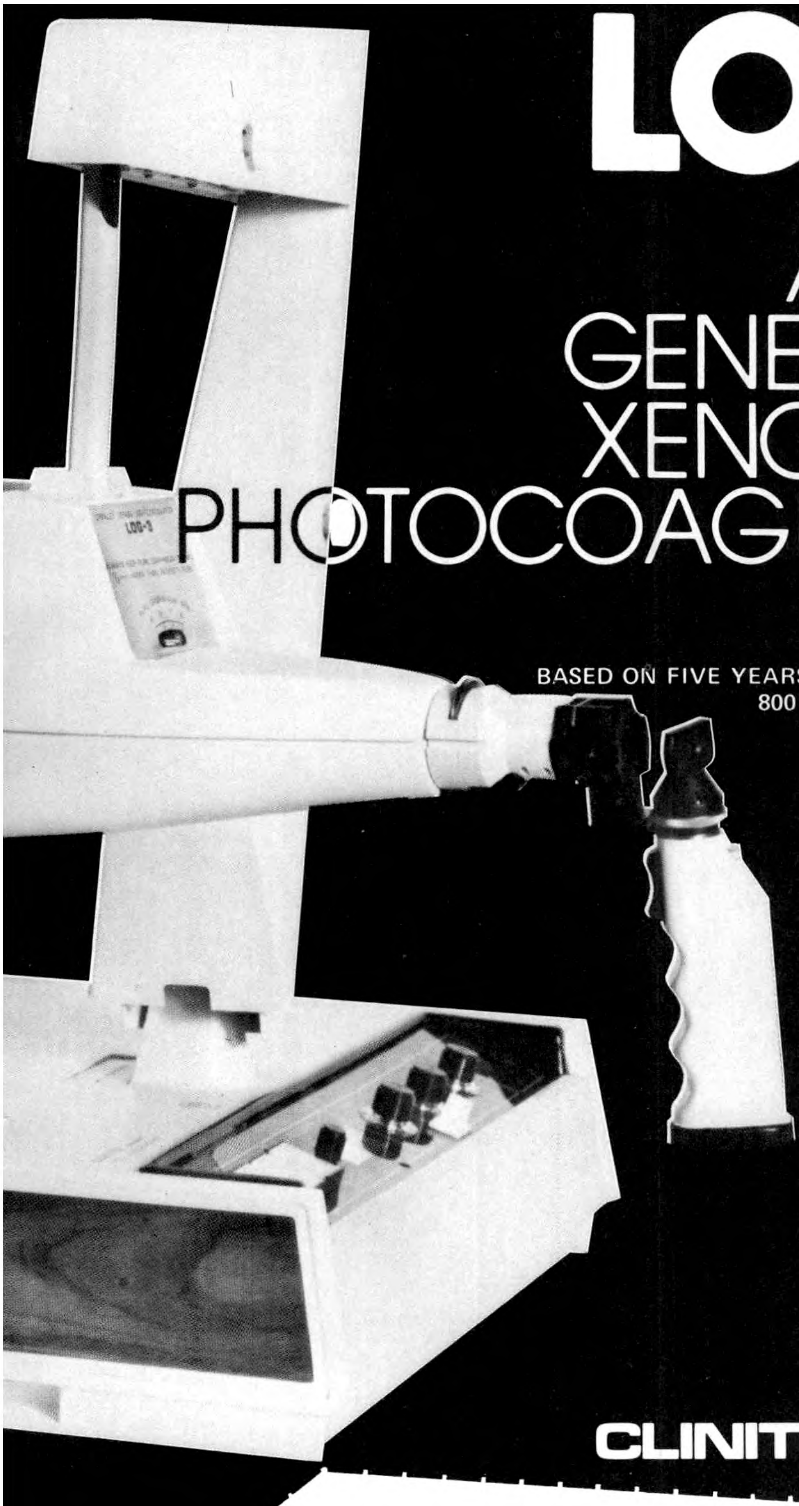

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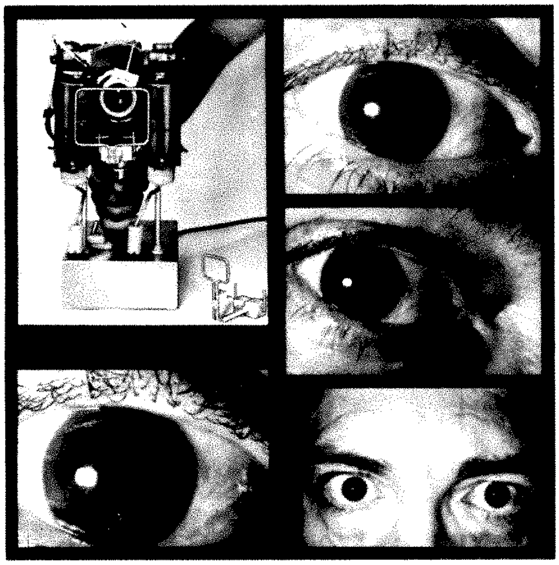

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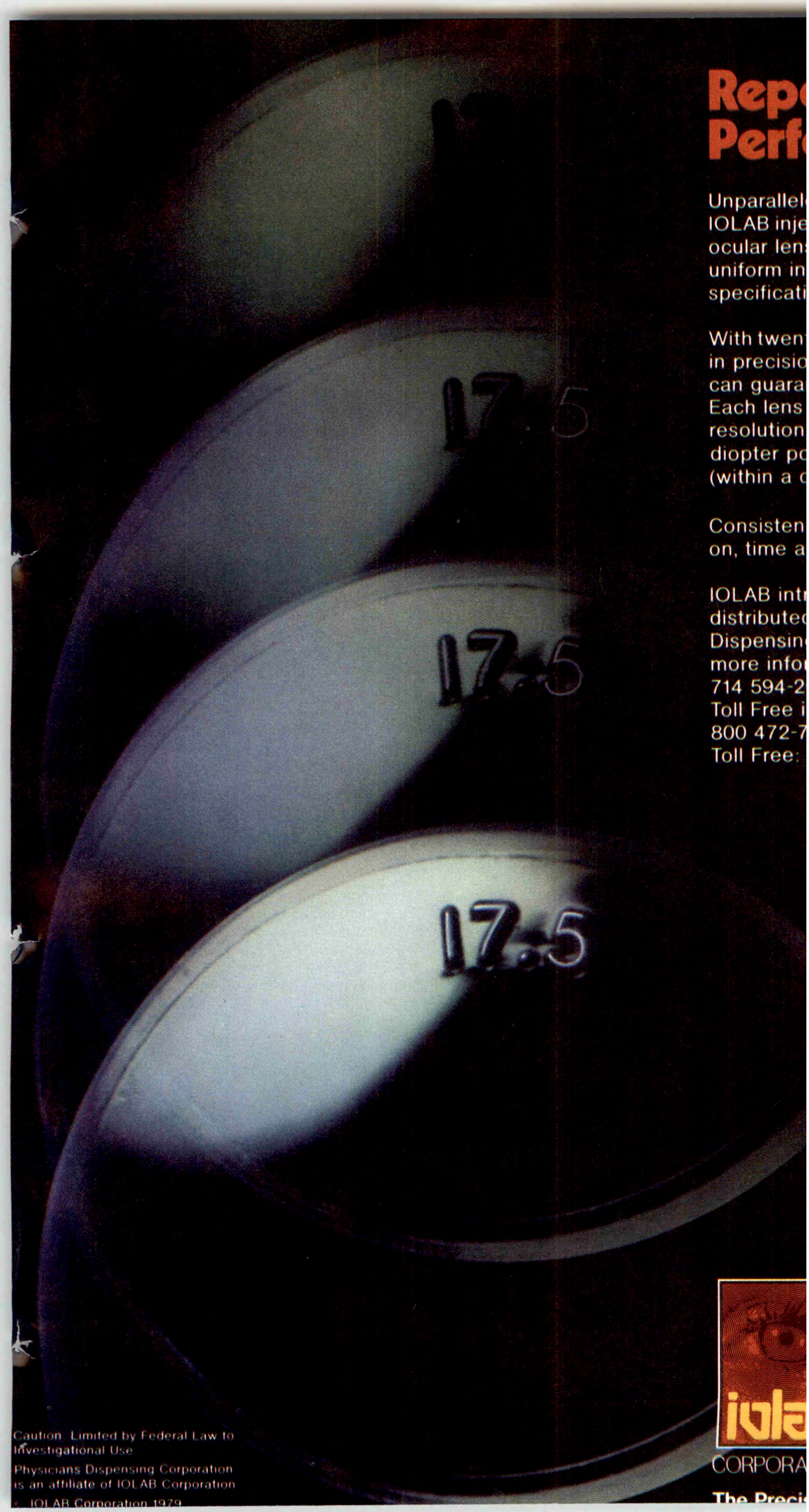

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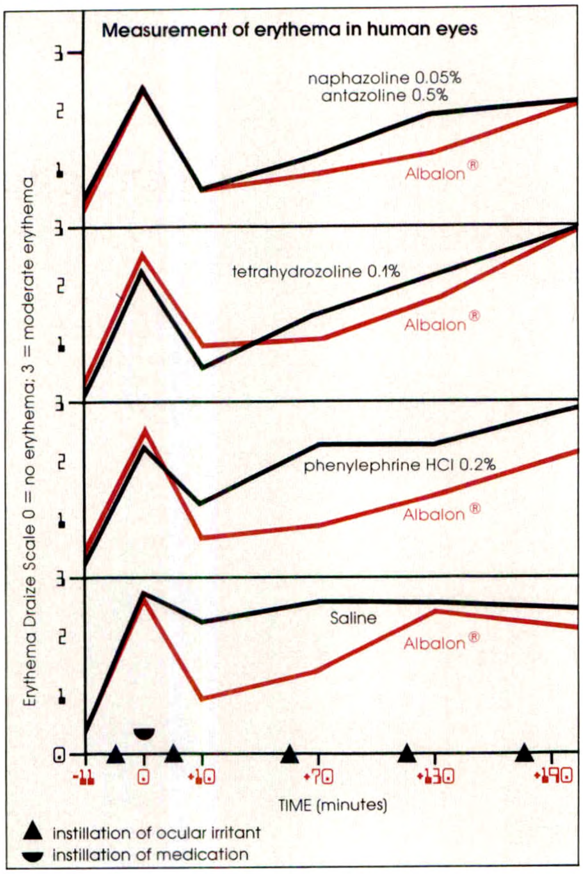

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 87 JANUARY, 1979 NUMBER 1

ALLERGIC GRANULOMATOUS NODULES OF THE EYELID AND CONJUNCTIVA

THE XXXV EDWARD JACKSON MEMORIAL LECTURE

NORMAN ASHTON, F.R.S., AND CHARLES COOK, F.R.C.S.

London, England

I deeply appreciate the honor of having been chosen to deliver the 35th Edward Jackson Memorial Lecture before the American Academy of Ophthalmology. It is an invitation I accepted with pleasure because, having recently retired from my position as Professor of Pathology at the Institute of Ophthalmology in London, it gave me an opportunity to thank my many American friends for their kindnesses through the years and also to declare to so many fellow members of the Academy my great admiration for the excellence of the science and practice of ophthalmology in America, so well exemplified in his time by Edward Jackson.

Clinically his eminence as an ophthalmologist was due especially to his important contributions to the art of refraction, rather a closed book to me, but with regard to his personal qualities I particularly admire the tribute paid to him by William Crisp in the First Jackson lecture:

> He personified the thought that the value of a man's life does not lie in the extent of his financial accumulations, but in the knowledge of work well done, of lessons well learned and handed on to others, in the richness of his experience and the sincerity of his purpose.

Truly on this testimony alone the life of Edward Jackson is worthy of our honor and respect.

I am grateful for the opportunity to round off my career with this distinction and to share it with my old friend and colleague Charles Cook, who worked with me in earlier days, by presenting a joint paper.

The subject I have chosen is an intriguing diagnostic problem in the conjunctiva, which I am sure ophthalmologists have encountered, but about which little has been written. I shall begin by describing a pathologic reaction that may not be familiar. It is known as the "Splendore-Hoeppli" phenomenon, and it refers to the histologic appearance of an amorphous eosinophilic material first described around fungus spores by Splendore in 1908.[1] He referred to it as asteroid formation and named the fungus *Sporotrichum asteroides*. A similar eosinophilic fringe was subsequently observed by Hoeppli[2] around *Schistosoma* eggs but, although he quoted an earlier reference,[3] he did not refer to Splendore. Hoeppli suggested that it represented a reaction between the ova and the surrounding tissue, perhaps through a secretion of the lateral glands, usually of the mature mira-

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From the Department of Pathology, Institute of Ophthalmology, University of London, England. This study was supported by a grant from the Leverhulme Trust. Presented before the annual meeting of the American Academy of Ophthalmology in Kansas City, Missouri, Oct. 22, 1978.

Reprint requests to Professor Norman Ashton, Royal College of Surgeons of England, 35/43 Lincoln's Inn Fields, London, WC2A 3PN, England.

cidium. This eosinophilic fringe was surrounded by necrotic material, nuclear remnants, belts of epithelioid cells, large mononuclear cells, giant cells, and many eosinophils. It is an eosinophilic granuloma with distinct histologic features. Lurie[4] subsequently reviewed the studies published up to 1963. The appearance of the eosinophilic hyaline fringe was further studied by von Lichtenberg and associates,[5-7] who demonstrated the precipitate around the *Schistosoma* eggs by using immunofluorescent techniques. They found the precipitate to be ova antigen and fixed host globulin that formed an antigen-antibody complex. This evidence convinced them that the Splendore-Hoeppli phenomenon was simply an in vivo antigen-antibody precipitate arising when certain immunologically critical conditions occur.

The electron microscopic appearance of this precipitate as seen around fungi was described by Williams and associates.[8] They found that it consisted of degenerating cytoplasmic and nuclear debris derived especially from eosinophils, but also from plasma cells, macrophages and lymphocytes, together with amorphous granular material, probably antigen-antibody complex, as described in the electron microscopy of the Arthus type of hypersensitivity.[9] A striking feature was the presence of eosinophils and Charcot-Leyden crystals, in their acicular and hexagonal forms, derived from eosinophils.[10-12] Williams and associates[8] noted the variability of the incidence of Splendore-Hoeppli precipitates in fungal infections and concluded that maximal host reactivity or hypersensitivity were essential for their formation by the in situ reaction of antigen antibody.

Gilbert, Khoury, and Pore,[13] in a study of the Splendore-Hoeppli phenomenon in fungal infection (*Entomophthora*), showed that the eosinophilic precipitate contained phospholipid, acid and neutral lipids, lipofuscin, diastase-resistant PAS-positive and PTAH-positive material, and neutral mucopolysaccharides. The precipitate stained red with Masson's trichrome stain but stained negatively with Ziehl-Neelsen's stain. These authors emphasized that the presence of the Splendore-Hoeppli phenomenon suggested *Entomophthora* infection.

I would like now to call attention to the Splendore-Hoeppli phenomenon as a nodular inflammatory lesion in the conjunctiva, episclera or eyelids, unilaterally or bilaterally, and usually not associated with lesions elsewhere. In a histologic study of 22 cases we found the causative agent in only four, and this proved to be a nematode, which could not be more specifically identified.

The histologic appearance of these ocular lesions is so characteristic of the Splendore-Hoeppli phenomenon, that even a quick glance at a section suggests this distinct entity. Of 22 cases listed in Tables 1 and 2, we have selected seven to illustrate the histologic findings and the diagnostic problems involved; in four the causative agent was found and in three no cause could be found.

CASE REPORTS

CASES OF KNOWN CAUSE

Case 1—A 14-year-old Ugandan girl had a subcutaneous swelling of the right upper eyelid of a few months' duration. No other abnormality was found apart from an eosinophilia of 18%. Sections showed several minute granulomas with granular eosinophilic centers containing basophilic nuclear debris, surrounded by epithelioid cells and giant cells, and more peripherally, by an inflammatory infiltration dominated by eosinophils (Fig. 1). In some eosinophils, cylindrical or irregular tubular hyaline structures were found with typical PAS-positive Splendore-Hoeppli precipitates surrounding the outer walls. These stained red with Masson stain (Fig. 2). Microdissection of the remaining tissue showed numerous Charcot-Leyden crystals (Fig. 3) and a fragment of what appeared to be a worm in a hyaline capsule, but this was inadequate material for further identification (Fig. 4). Electron micrographs taken from a selected area of the paraffin

TABLE 1

CASES OF KNOWN PARASITIC CAUSE*

Case No., Sex, Age (yrs)	Country	Clinical History	Histologic Findings	Laboratory Findings
1, F, 14	Uganda	Right upper eyelid; subcutaneous swelling; few months' duration; no other abnormality	Several minute granulomas with granular eosinophilic centers containing nuclear debris surrounded by epithelioid cells, giant cells, and eosinophils; worm fragments found in some lesions (44 μ wide); Splendore-Hoeppli precipitates present; microdissection: fragment of a worm found in hyaline capsule; insufficient for identification	Blood eosinophilia 18%; electron microscopy showed typical structure of a nematode
2, M, 6	Uganda	Left upper eyelid; tumor of 3 wks' duration; eyelid swollen and painless; nodules in upper eyelid levator muscle	Granuloma containing segments of a compressed worm (14 μ wide), surrounded by giant cells; eosinophils ++; IgG, IgM, and C_3 positive in worm fragments and around (horseradish peroxidase)	Electron microscopy showed typical cuticle of a nematode worm together with Splendore-Hoeppli precipitate; no further identification possible
3, F, 34	U.K. (London)	Left conjunctiva; subconjunctival nodule at 9 o'clock, of 2 wks' duration; ? phlycten; no other abnormality	Granuloma with central eosinophilic material containing nuclear debris surrounded by epithelioid cells, giant cells and eosinophils + + +; resectioned 16 yrs later; fragments of a worm were found (62 μ wide)	Immunofluorescent tests with *Toxocara* antisera on the paraffin section was negative; electron microscopy showed the typical structure of a nematode and the ultrastructure of the Splendore-Hoeppli phenomenon
4, M, 28	Uganda	Right conjunctiva; subconjunctival nodule of "short duration" in upper temporal quadrant	Inflammatory fibrous tissue containing a necrotic eosinophilic focus in which several worm fragments were found (approximately 55-82 μ wide); eosinophils +	

*Mild, +; moderate, ++; and severe, +++; ? signifies doubtful.

TABLE 2

CASES OF UNKNOWN CAUSE*

Case No., Sex, Age (yrs)	Country	Clinical History	Histologic Findings	Laboratory Findings
5, M, 2	U.K. (Hereford)	Bilateral conjunctiva; nodular swelling at 12 o'clock near corneoscleral limbus; ? phlyctenules or nevi; premature birth and anemic; no other significant abnormality; chest X-ray normal; lesions disappeared but 7 mos later recurred in left eye; 9 mos later in both eyes resembling phlyctenular conjunctivitis; 23 mos later further recurrence which disappeared; 8 yrs later patient in excellent health	Two subepithelial nodules with a central eosinophilic mass containing nuclear debris and surrounded by epithelioid cells and giant cells ingesting eosinophilic material; eosinophils +; no etiologic agent found	Electron microscopy showed the ultrastructure of the Splendore-Hoeppli phenomenon: electron-lucent and electron-dense material, cellular debris, and Charcot-Leyden crystals; no causative agent found
6, M, 5	U.K. (Hereford)	Right conjunctiva; a few yellow episcleral nodules in the upper half of the eye, no other abnormality; history of allergy to dogs; patient never out of U.K.; biopsy specimen taken; 5 mos later similar nodules appeared in the superior temporal quadrant of left eye; biopsy specimen taken; 1 mo later another "lump appeared but dispersed rapidly with drops"	First biopsy showed a granuloma with the appearance of the Splendore-Hoeppli phenomenon; serial sections revealed no causative agent; second biopsy showed several subepithelial conjunctival nodules with an exactly comparable appearance but no organisms of any kind were found in over 500 serial sections	White blood cell count normal on two occasions; *Toxocara* fluorescent antibody test negative; feces negative for parasites; electron microscopy showed featureless debris and Charcot-Leyden crystals
7, F, 23	U.K. (London)	Left conjunctiva; small lump which appeared during an attack of herpetic conjunctivitis (antiviral therapy); not known to have been abroad; no further information	Minute granuloma with necrotic eosinophilic center and surrounding epithelioid cells and a few giant cells; eosinophils +; no causative agent found	
8, M, 3	U.K. (Middlesex)	Conjunctiva; small yellow nodule of the conjunctiva 5 mm from corneal margin in the upper nasal quadrant; no other abnormality; never been abroad; no history of allergy; 3 yrs later patient in good health	Two minute granulomas with the same structure as described above; eosinophils and giant cells, however, were more prevalent; no causative agent found	

9, M, 2½	U.K. (Kent)	Left conjunctiva; yellowish vascular nodule in upper nasal quadrant, completely excised; similar lesion appeared shortly thereafter in the right eye, resolved of its own accord; recurred 9 mos later and again at 11 mos; the lesions resolving with betamethasone (Betnesol) drops; no allergies; never been abroad; now in good health	Isolated sharply demarcated granulomatous lesions with intensely eosinophilic center containing cellular debris, engulfed by multinucleated giant cells all surrounded by epithelioid cells and scattered eosinophils and mononuclear cells; eosinophilic center stains red with Masson 44/41; fading into an intense blue-black peripherally; no causative agent found in over 90 serial sections	White blood cell count, 8,600/mm^3 (4% eosinophils); no evidence of parasites in feces or blood smear; electron microscopy (piece taken from paraffin block) showed cellular debris: granular and homogenous deposits, some engulfed in mononuclear cells, suggestive of proteinaceous deposits (?) immune complexes; no causative agent found
10, M, 7	U.K. (Essex)	Right conjunctiva; small limbal lesion with edema at 11 o'clock, one month. Became raised 2 wks later; ? phlycten; excised; recurrence of inflammation 15 mos later; no allergies; patient was in India 1970-1971; now in good health	Area of eosinophilic structureless material surrounded by giant cells with plasma cells and eosinophils; hyaline fragments present; ? foreign body; ? nature; no causative agent identified	
11, M, 4	U.K. (London)	Right conjunctiva; slowly growing yellowish nodule; few months' duration; general health good; no allergies; no recurrence; had not been abroad	Two or three foci of eosinophilic material surrounded by giant cells and eosinophils; no causative agent found	
12, M, 25	U.K. (Middlesex)	Bilateral conjunctiva; rapid onset of bilateral conjunctivitis which subsided within 2 days leaving many inflamed conjunctival nodules, these developed during an exacerbation of asthma (similar buccal mucosal lesions were also present but no biopsy was done); 18 mos later recurrent granulomas of the conjunctiva appeared associated with a swelling of the temporal muscles and subcutaneous mobile nodules in the outer canthal area; the patient is an asthmatic and has an asthmatic family	Conjunctival sections show focal areas of necrotic eosinophilic debris containing nuclear fragments and surrounded by giant cells and inflammatory cells, including many eosinophils; the findings corresponded exactly to our other cases in which a worm was found and further sections confirmed the staining reactions of Splendore-Hoeppli phenomenon; no causative agent was found; section of temporal muscle showed edema and dense infiltration with eosinophils; no causative agent found;	Eosinophils 20%; no microorganisms or parasites found and appropriate CFTs were negative; ESR 1 mm/hr; RPCFT, CWR, and VDRL, negative; no fungi or organisms found in biopsy material; no ova in rectal snips; red blood cells ++ in feces; *Toxocara* skin test, positive but fluorescent antibody test (*Toxocara*) was negative; chest X-ray, negative; sputum, no fungi; Mantoux 1/10,000, negative; blood biochemistry normal except for uric acid (7.1 mg/100 ml); toxoplasmosis dye test, negative

Table Continued

TABLE 2 (*Continued*)

CASES OF UNKNOWN CAUSE*

Case No., Sex, Age (yrs)	Country	Clinical History	Histologic Findings	Laboratory Findings
		history; he is allergic to penicillin and skin tests show allergy to pollen, fur, and house dust; follow-up: 4 yrs after onset the patient is in good health apart from asthma controlled with prednisolone; except for one small evanescent conjunctival lesion, no recurrence of ocular condition	section of teased nodule of outer canthus showed areas of eosinophilic debris similar to conjunctiva; no causative agent found	
13, M, 5	Jerusalem	Conjunctiva; white episcleral nodule (? caused by worm)	Irregular nodule of partially calcified, eosinophilic hyaline material; some giant cells, eosinophils, and epithelioid cells surround the hyaline foci; no causative agent found	
14, M, 4½	Jerusalem	Conjunctiva; small conjunctival cyst; vernal catarrh	A localized focus of eosinophilic degeneration (red with Masson 44/41 stain) containing bacilliform and thread-like strands of nucleoplasm surrounded by giant cells, epithelioid cells, and eosinophils; no causative agent found in serial sections	
15, M, 13	Jerusalem	Conjunctivoscleral nodule 12 o'clock (? caused by worm)	Eosinophilic mass of necrotic tissue surrounded by giant cells, epithelioid cells, and inflammatory cells; eosinophils ++; no causative agent found	
16, M, 18	Jerusalem	Conjunctiva. Localized noninflammatory swelling of 2 wks' duration (? parasite)	Small focus of granular eosinophilic material engulfed by giant cells, epithelioid cells, and eosinophils; serial sections; no causative agent found	
17, M, 5	Jerusalem	Bilateral conjunctiva; discrete nodules scattered over upper halves of both conjunctivae; 1½ mos' duration; biopsy	Small focus of necrotic eosinophilic material with nuclear debris surrounded by giant cells, epithelioid cells, and eosinophils ++; no	

18, F, 20 mos	Jerusalem	Conjunctiva; red eye of 1 wk's duration; biopsy from superior limbal area	Scattered circumscribed eosinophilic necrotic foci with giant cells and eosinophils (Masson 44/41, central foci stains dark blue with points of red with Picro-Mallory scarlet); one focus showed swollen eosinophilic collagen fibers traversing the lesion; serial sections: (100) no causative agent found	
19, M, 3	Jerusalem	Left conjunctiva; small subconjunctival nodules adherent to sclera in upper half; two wks' duration; other eye normal; no other abnormality; no recurrence	Several granulomatous foci consisting of eosinophilic material containing irregular bacilliform chromatin fragments, surrounded by histiocytes, giant cells, inflammatory cells, and eosinophils. The giant cells have ingested eosinophilic material and some of the granulomata are traversed by swollen collagen fibers; serial sections: no causative agent found	Electron microscopy showed amorphous debris only
20, M, 27	Uganda	Left conjunctiva; after 5 days of swelling and redness a sausage-shaped nodule appeared in temporal half of eye	Sharply localized eosinophilic foci with nuclear debris surrounded by giant cells and histiocytes; eosinophils +; serial sections: no causative agent found	
21, F, 24	Uganda	Right conjunctiva; subconjunctival sausage-like swelling in upper half of eye; a whitish catgut-like thread could be seen extending towards upper rectus muscle; 1 mo history; moderate exophthalmos; ? parasite	Several sharply demarcated granulomas showing the exact appearance previously described; serial sections: no causative agent found	Blood: eosinophilia 61%; stools: ankylostoma positive
22, M, 6	Sudan (Khartoum)	Left conjunctiva; multiple nodules mainly in the interpalpebral zone present for 3-4 wks; eight on nasal side; four on temporal side, about 0.5 mm in diameter (ophthalmologist stated that he had seen five similar cases which cleared up spontaneously)	Multiple small eosinophilic necrotic foci surrounded by giant cells, histiocytes, and eosinophils; no causative agent found	

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*CFT indicates complement fixation test; ESR, erythrocyte sedimentation rate; RPCFT, Reiter protein complement fixation test; CWR, Cardiolipin Wassermann reaction. Mild, +; moderate, ++; severe, +++; ? designates doubtful.

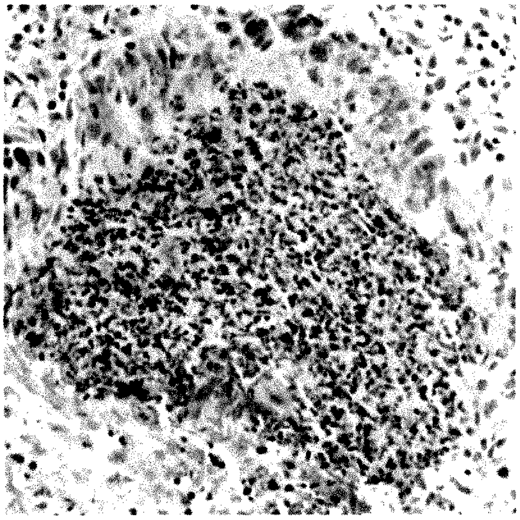

Fig. 1 (Ashton and Cook). Case 1. Section of eyelid. Minute granuloma with eosinophilic center containing nuclear debris surrounded by epithelioid cells, giant cells, and eosinophils (hematoxylin and eosin, ×280).

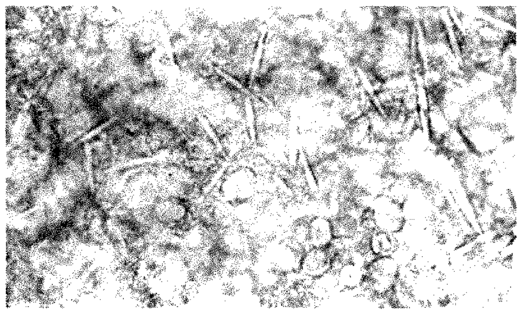

Fig. 3 (Ashton and Cook). Case 1. Wet preparation of granuloma showing cellular debris and numerous Charcot-Leyden crystals (×720).

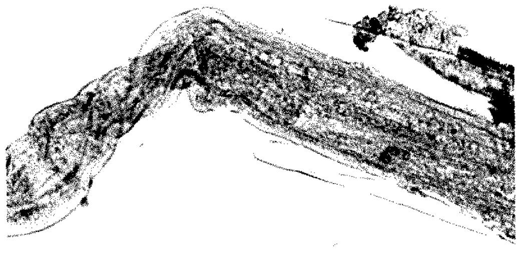

Fig. 4 (Ashton and Cook). Case 1. Structure isolated from granuloma by microdissection appears to be a worm ensheathed in its moulting cuticle (×225).

section showed the typical structure of a nematode (Fig. 5) (compare with Bird[14]).

Case 2—A 6-year-old Ugandan boy had a three-week history of a painless swollen left eye, caused by a tumor of the left upper eyelid. The lesion was excised and some nodular patches were also found in the fascia of the upper eyelid levator muscle.

Sections showed a granulomatous mass with cystic spaces surrounded by giant cells, and segments of a degenerating worm (Fig. 6). In a longitudinal section the Splendore-Hoeppli precipitate was seen (Masson stain red) (Fig. 7). Numerous eosinophils were found in the surrounding tissue.

Electron micrographs taken from a selected area of the paraffin section again revealed the typical cuticle of a nematode, together with the amorphous Splendore-Hoeppli precipitate on its surface (Fig. 8). No further identification could be made.

Case 3—A 34-year-old woman from London had a painful phlycten-like nodule of two weeks' duration at 9 o'clock in the bulbar conjunctiva of the left eye. No other abnormality was found. At operation the nodule was removed, and was found to be in Tenon's capsule and not attached to the sclera.

Sections showed the typical appearances of a granuloma with an eosinophilic center of nuclear debris surrounded by granulomatous tissue with eosinophils and giant cells (Fig. 9). The patient was subsequently lost to follow-up.

This case was originally reported in 1961 as an episcleral granuloma of unknown cause, but because of the present investigation the sections were re-examined and the Splendore-Hoeppli reaction recognized. Serial sections were then taken from the block and fragments of what appeared to be a worm were discovered within the granuloma (Fig. 10). Electron micrographs taken from a selected area of the paraffin section showed the typical structure of a nematode lying in a Splendore-Hoeppli precipitate (Figs. 11-13). Thus, the correct diagnosis was reached 16 years after the original biopsy had been done, but the patient could not be traced and whether she had been out of the country is unknown.

Case 4—A 28-year-old Ugandan man had a subconjunctival thread-like nodule in the upper temporal quadrant of short duration. The lesion was excised. Sections showed inflammatory fibrous tissue that had a necrotic eosinophilic focus with a surrounding granulomatous reaction containing giant cells and many eosinophils. Within this tissue several worm fragments were found (55 to 82 μ wide) (Fig. 14). Electron micrographs taken from a selected area of the paraffin section showed the typical cuticle of a nematode with transverse annulations and oblique cuticular tubules (Fig. 15).

CASES OF UNKNOWN CAUSE

Case 5—A 2-year-old English boy was brought to us because of blisters in both eyes of two days' duration. Examination revealed solitary small pigmented cyst-like swellings symmetrically placed

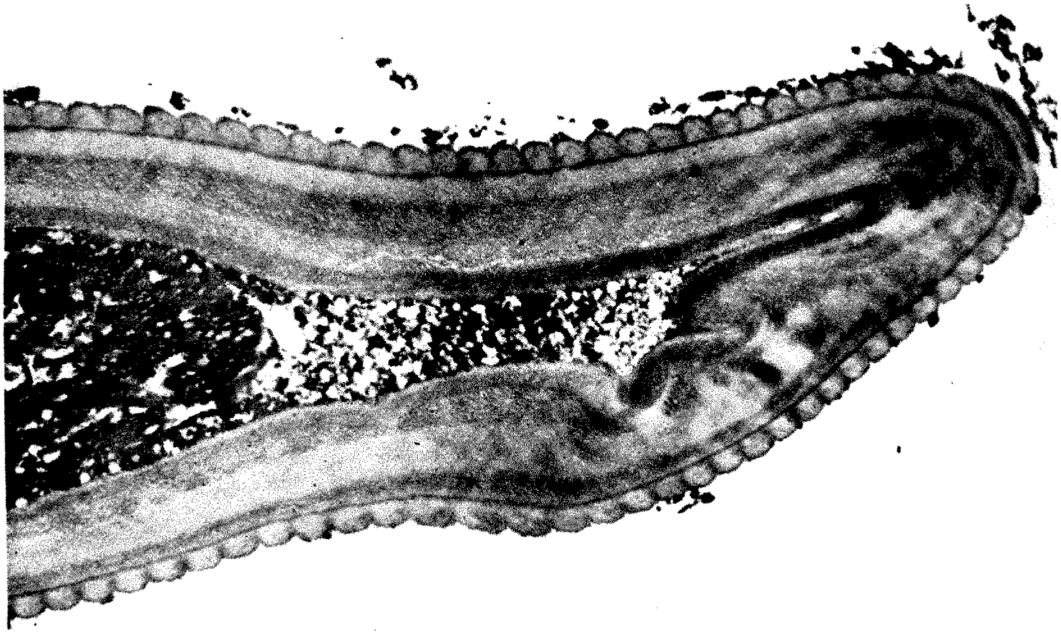

Fig. 5 (Ashton and Cook). Case 1. Presumed worm fragment in granuloma shows the cuticle with transverse annulations typical of a nematode (electron micrograph, ×9,900).

near the corneoscleral limbus at 12 o'clock in both eyes with associated areas of injection. These were thought to be phlyctenules or possibly nevi. There were two small nodules in the skin of the abdomen with palpable glands in the groins that were considered to be within normal limits. There was no history of injury or other eye trouble. Birth was premature (weight 1.8 kg) and the boy was being treated for anemia (hemaglobin, 51%). He had never been overseas and no animals or birds were kept in the house. A chest x-ray was normal and general examination by a pediatrician revealed no abnormality. The child was treated with corticosteroid drops, but one month later he developed two nodules on the right conjunctiva and a biopsy was performed.

Sections were similar in appearance to those already described. There were two subepithelial nodules, lying in a mildly inflamed conjunctival stroma

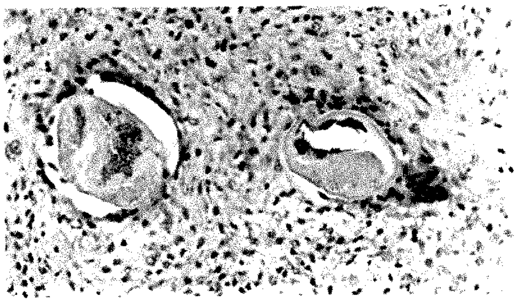

Fig. 6 (Ashton and Cook). Case 2. Eyelid granuloma containing transverse segments presumably of a worm (hematoxylin and eosin, ×225).

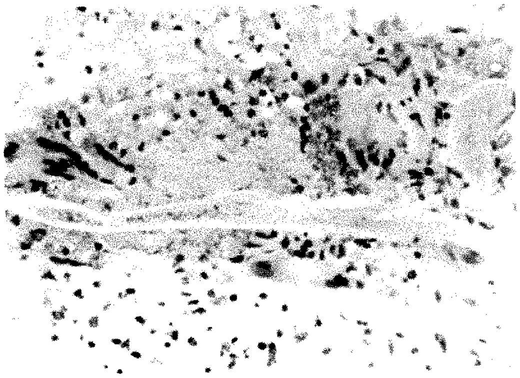

Fig. 7 (Ashton and Cook). Case 2. Granuloma containing longitudinal fragment of presumed compressed worm. Note the surrounding eosinophilic Splendore-Hoeppli precipitate and giant cells (hematoxylin and eosin, ×560).

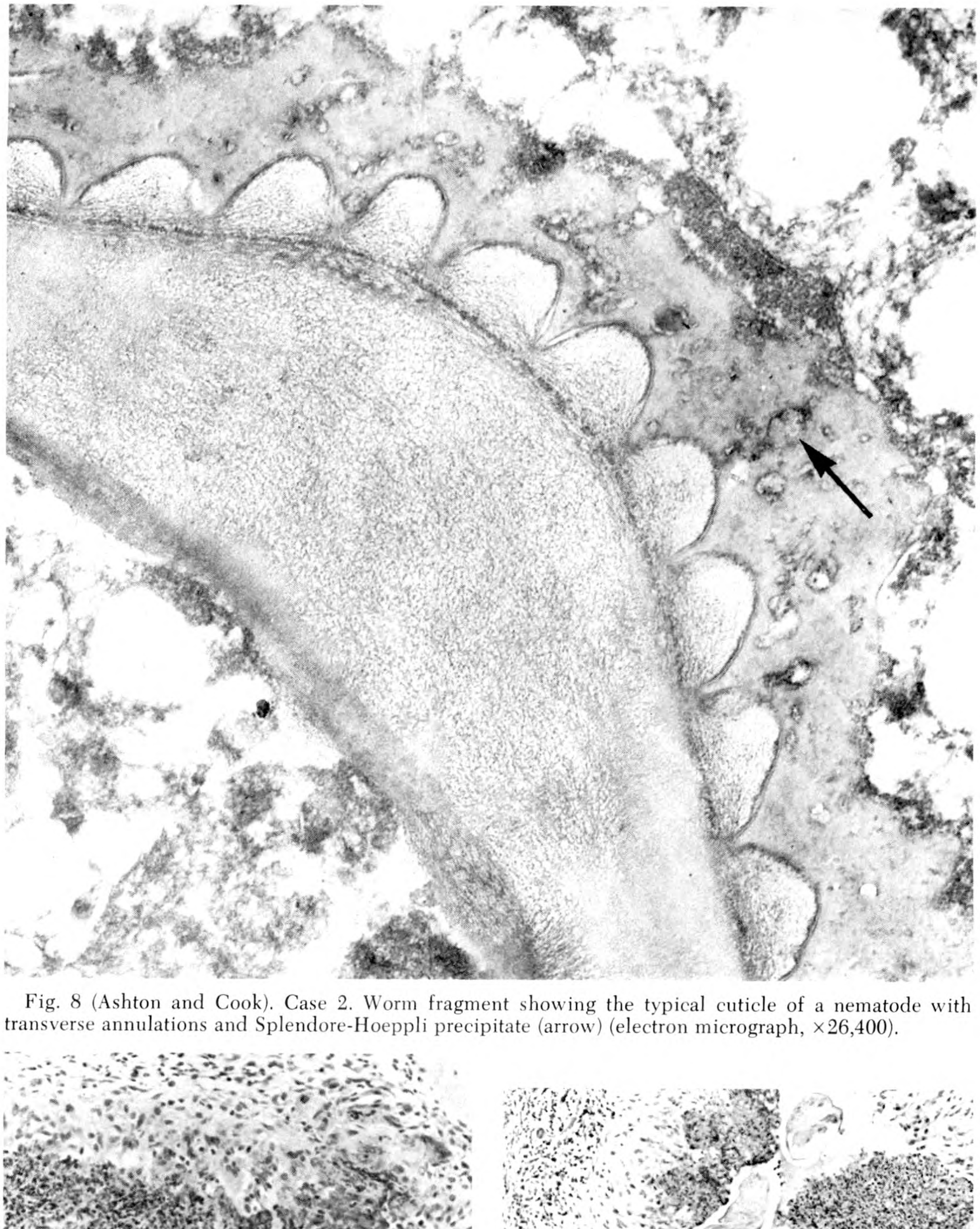

Fig. 8 (Ashton and Cook). Case 2. Worm fragment showing the typical cuticle of a nematode with transverse annulations and Splendore-Hoeppli precipitate (arrow) (electron micrograph, ×26,400).

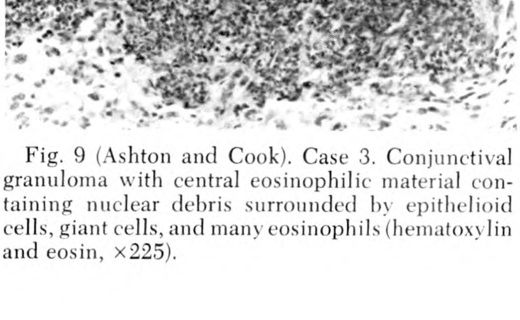

Fig. 9 (Ashton and Cook). Case 3. Conjunctival granuloma with central eosinophilic material containing nuclear debris surrounded by epithelioid cells, giant cells, and many eosinophils (hematoxylin and eosin, ×225).

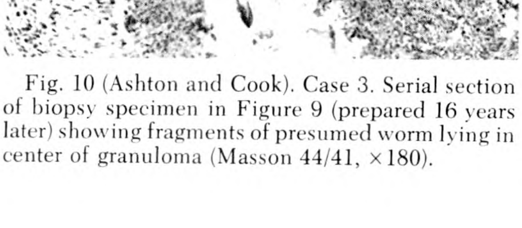

Fig. 10 (Ashton and Cook). Case 3. Serial section of biopsy specimen in Figure 9 (prepared 16 years later) showing fragments of presumed worm lying in center of granuloma (Masson 44/41, ×180).

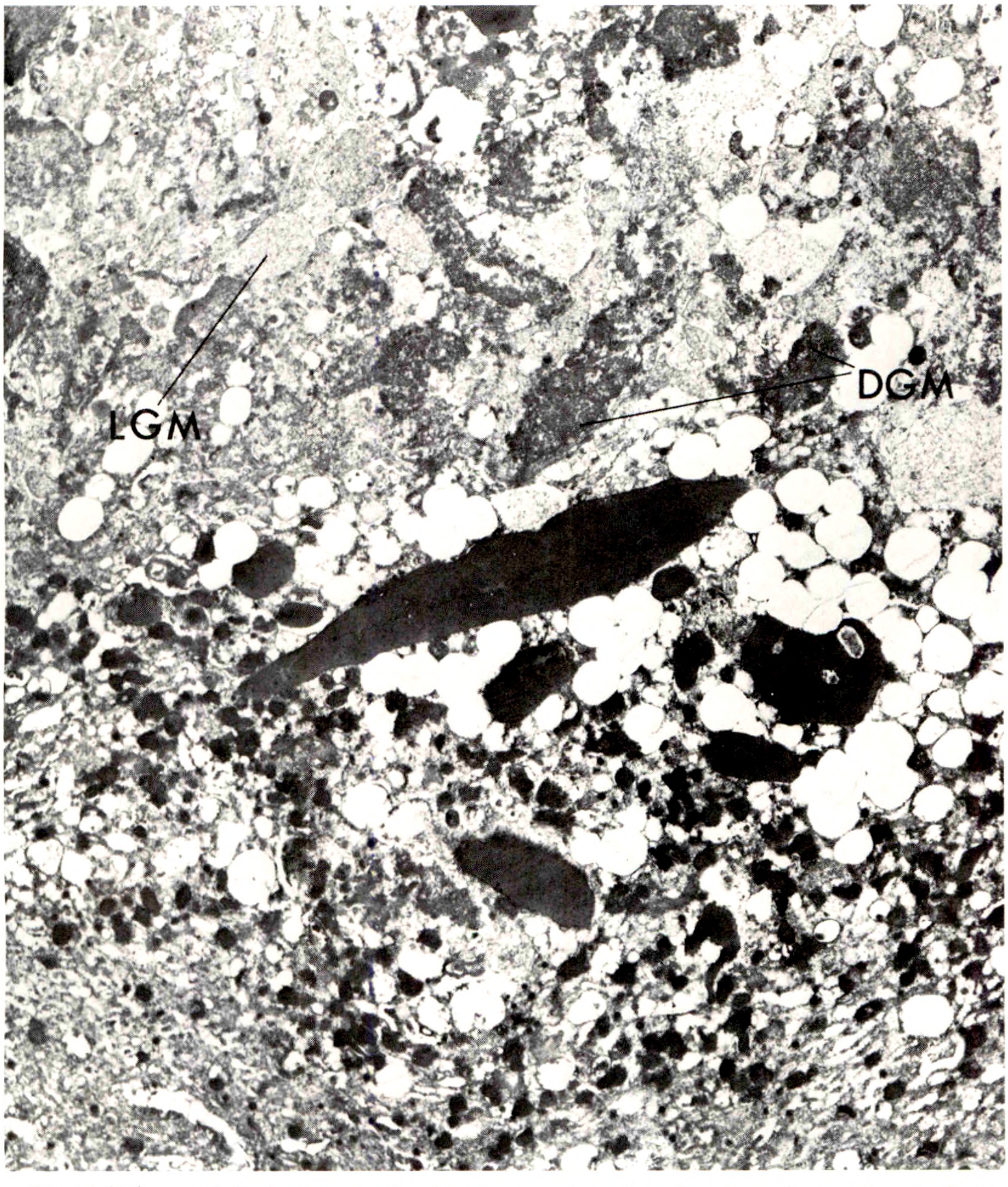

Fig. 11 (Ashton and Cook). Case 3. The ultrastructure of the Splendore-Hoeppli precipitate is shown. Numerous Charcot-Leyden crystals, eosinophil granules, cellular debris, and electron-dense (DGM) and translucent (LGM) granular material are seen (electron micrograph, × 6,000).

that had a central eosinophilic mass containing basophilic nuclear debris, surrounded by epithelioid cells, and giant cells ingesting eosinophilic material. Only a moderate number of eosinophils were present (Fig. 16). Serial sections and special stains failed to reveal any etiologic agent.

Subsequently, the lesions disappeared but recurred in the left eye after seven months and in both eyes at nine months (situated near the corneoscleral limbus at 1 to 2 o'clock and resembling phlyctenular conjunctivitis), and again after 23 months. No further biopsies were performed.

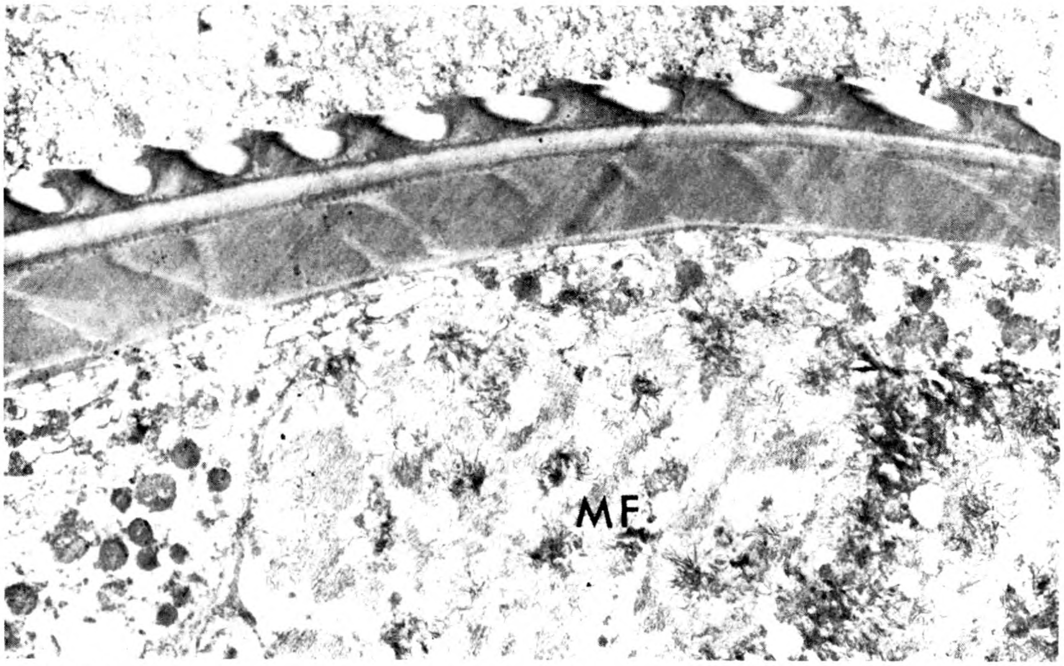

Fig. 12 (Ashton and Cook). Case 3. Worm fragment shows the cuticle of a nematode with pronounced transverse annulations, a clear internal cortical layer, and a wide basal layer with oblique striations. Muscle fibers (MF) are seen in the hypodermis (electron micrograph, ×5,900).

The patient was recalled for examination eight years later and was found to be in excellent health with no ocular abnormality. Electron micrographs taken from a selected area of the paraffin section showed the ultrastructure of the Splendore-Hoeppli phenomenon with electron-dense and electron-lucent granular material, cellular debris, and Charcot-Leyden crystals (Figs. 17 and 18). No causative organism was found.

Case 6—A 5-year-old English boy complained of an inflamed right eye in March 1973. Examination revealed a few yellow episcleral nodules in the upper half of the right eye. There was no other abnormality except for a past history of allergy to dogs, which caused swelling of the eyes. The boy had never been out of England. A differential white blood cell count was normal. A biopsy was performed in April 1973, and by June 1973 the eye was normal. In August 1973, similar nodules appeared in the episclera in the superior temporal quadrant of the left eye (Fig. 19). Three pieces were removed for biopsy in September 1973. The white blood cell count was repeated and found to be normal. Stool examination showed no evidence of ova or parasites. The mother reported that in September 1973 another lump appeared but disappeared rapidly with eye drops; the patient is now well.

Sections from the first biopsy showed that the nodule consisted of a mass of eosinophilic material with nuclear debris (Feulgen-positive) and amorphous basophilic material apparently composed of lysed nuclei, and surrounded by epithelioid cells and a moderate number of eosinophils (Fig. 20). The eosinophilic material stained red with Masson trichrome stain and Masson 44/41 stain; blue with PTAH. The appearance was that of the Splendore-Hoeppli phenomenon and a worm was suggested as the possible causal agent. Serial sections were cut through the lesion but no worm or other organism was found. Electron micrographs taken from a paraffin section showed structureless debris and Charcot-Leyden crystals only.

Sections from the second biopsy showed several subepithelial conjunctival nodules exactly like those of the first biopsy. More than 500 serial sections were cut through the tissues but no organism was found.

Case 12—A 25-year-old English man had suffered with asthma for one year and in January 1973 had conjunctival edema. Five weeks previously he had developed bilateral conjunctival swelling and redness with some itching and a mild watery discharge from his right eye. These symptoms came on rapidly within about 12 hours. He developed a little swelling of the eyelids. Visual acuity remained unaffected. The inflammation and swelling subsided in one

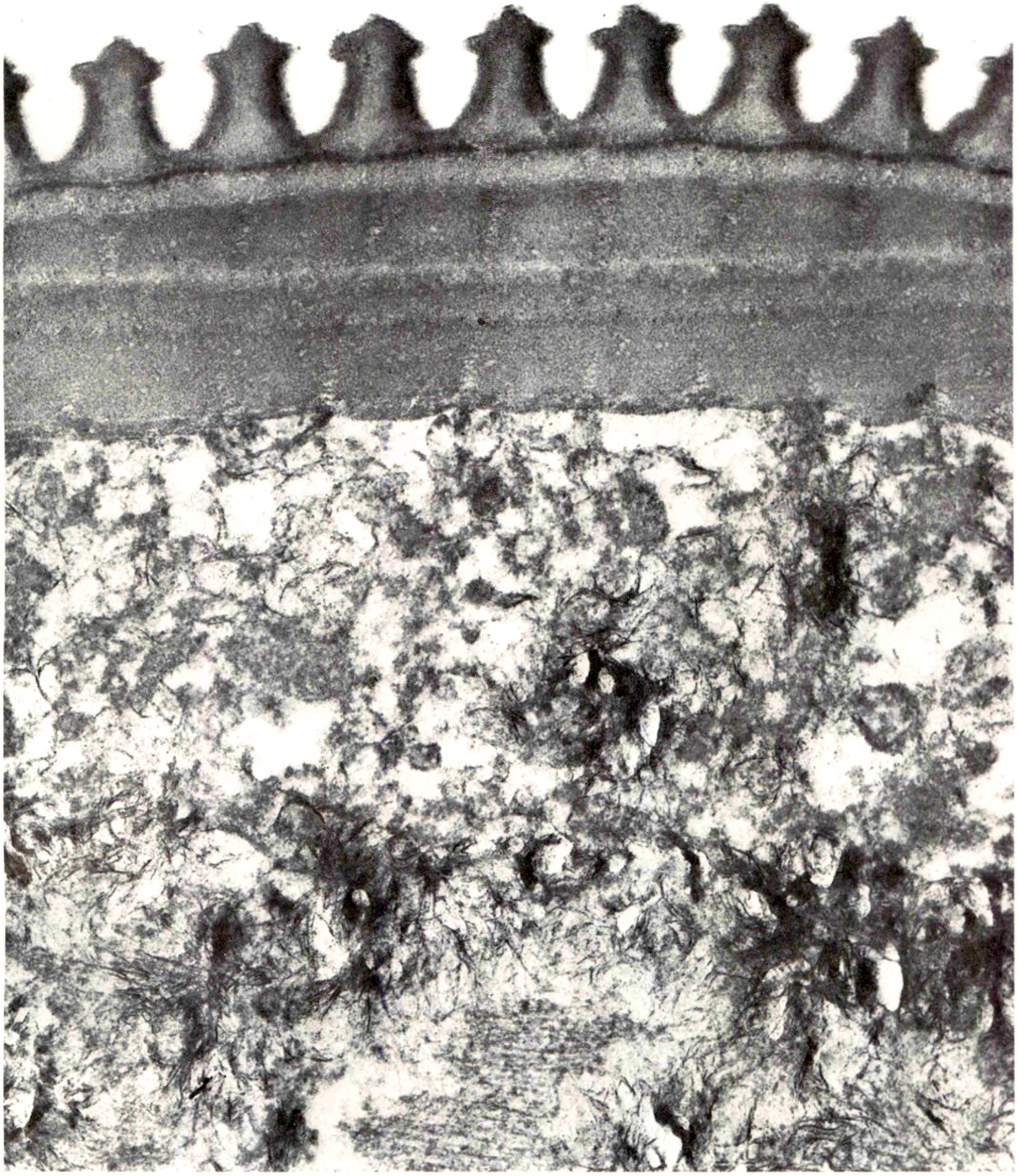

Fig. 13 (Ashton and Cook). Case 3. Higher power view of similar area shown in Figure 12 (electron micrograph, × 30,000).

or two days, leaving a number of inflamed nodules, which disappeared in the next week or so; asthma was his only complaint. His ocular abnormality, which developed during exacerbation of his asthma, was associated with withdrawal of corticosteroid therapy. At about the same time he developed swellings inside his mouth and on the gums, and these similarly subsided; he then developed lesions at the back of the mouth. He had spent two weeks in Israel, Greece, and Turkey in 1967; Italy in 1968,

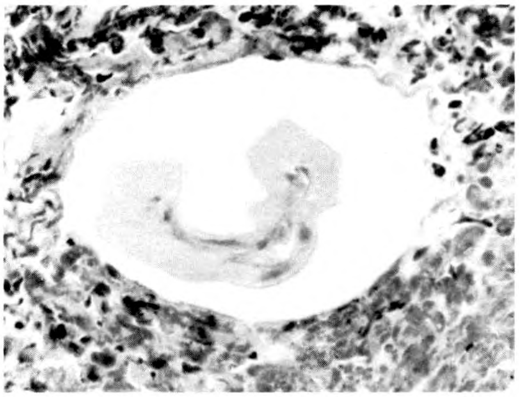

Fig. 14 (Ashton and Cook). Case 4. Section of conjunctiva. A chronic eosinophilic inflammatory focus shows a fragment of presumed worm (hematoxylin and eosin, ×450).

Spain in 1971, and North Africa 12 years ago. He had no history of contact with cats or dogs. He was allergic to penicillin and reacted to pollen, house dust, and fur.

On examination he had a series of scattered nodules in the bulbar subconjunctival tissue of both eyes, that appeared as either small rounded elevations about 2 mm in diameter, or as larger confluent masses. A striking feature was the presence of creamy deposits in most of them, forming either short branching linear shapes or confluent masses (Fig. 21). One or two of the smaller lesions were ulcerating through the conjunctiva, and were surrounded by a dense cellular infiltration and intense vascular dilation.

Despite careful search with the slit-lamp microscope no identifiable objects could be found in the lesions. They did not appear to be phlyctenular nodules because there was no necrosis of the overlying conjunctiva and they were painless and not tender. The confluent masses, especially in the outer part of the bulbar conjunctiva toward the outer fornix, were completely dissimilar from phlyctenular disease, and the cornea was not involved. The lesions were not typical of sarcoidosis. A biopsy specimen was taken from the right eye.

Sections showed within the subconjunctival tissue focal areas of necrotic eosinophilic debris with nuclear fragments surrounded by giant cells and inflammatory cells, including a conspicuous number of eosinophils in the adjacent stroma (Fig. 22). These histologic features corresponded exactly with those cases in which we had found a nematode and, coupled with the blood eosinophilia and increased IgE, we thought the patient's condition may have been caused by helminthiasis. Further biopsy specimens were taken for sectioning, microdissection, and electron microscopy. The sections confirmed the earlier findings: special stains showed the tinctorial characteristics of Splendore-Hoeppli precipitates in the center of the nodules (Charcot-Leyden crystals stained bright red with rhodamine B). No parasites, fungi, or other organisms could be found in 100 serial sections by microdissection or electron microscopy. Electron micrographs showed electron-dense material consisting of aggregations of irregular cystic structures, lying in a less dense amorphous granular mass, in which fragments of collagen, nuclei, degenerating cytoplasmic contents, lipid globules, eosinophilic granules, Charcot-Leyden crystals and fibrillar deposits of fibrin were seen (Figs. 23-25). No parasitic or other causative agent could be identified.

Further biopsy specimens were taken for sectioning, microdissection, and electron microscopy; 450 serial sections were examined from one block and 80 from a nodule of the outer canthus, but without elucidation. About eight months after the patient's first examination he developed recurrent granulomas of the conjunctiva and swellings on both cheeks; biopsy specimens were taken of the right facial and temporal muscles.

Sections showed no abnormality in the facial muscle but the temporal muscle was edematous and densely infiltrated with inflammatory cells consisting predominantly of eosinophils. No causative agent was found in 75 serial sections.

A series of blood cell counts disclosed a variable eosinophilia, which at one stage reached 20% (2,200 cm^3). Erythrocyte sedimentation rate was normal and blood pressure was 130/80 mm Hg. He was referred to the Hospital for Tropical Diseases where they found no evidence of any active parasitic infection. He had *Schistosoma* and filaria complement fixation tests; day and night smears for microfilariae; sputum; urine and stool microscopy and culture; and rectal snips. Immunoelectrophoresis revealed normal IgG and IgA; increased IgM (256 IU/ml); C-reactive protein was not present; radio-immunoassay revealed IgE, 1,000 μ/ml (normal, 250 to 500 μ/ml). The toxocaral skin test was positive but the fluorescent antibody test was negative. Toxocariasis was not thought to be the cause of his current condition.

On follow-up four years later in December 1977 the patient was found to be in good health although still subject to asthmatic attacks, which were controlled by prednisolone. Except for one small conjunctival lesion that rapidly disappeared there had been no recurrence of his ocular condition.

DISCUSSION

Of the 22 patients in our study 17 were male and five were female. Sixteen were less than 18 years old; of these, 13 were less than 8 years old. Thus, the condition was commonest in boys. In 18 cases (including the four cases shown to be caused

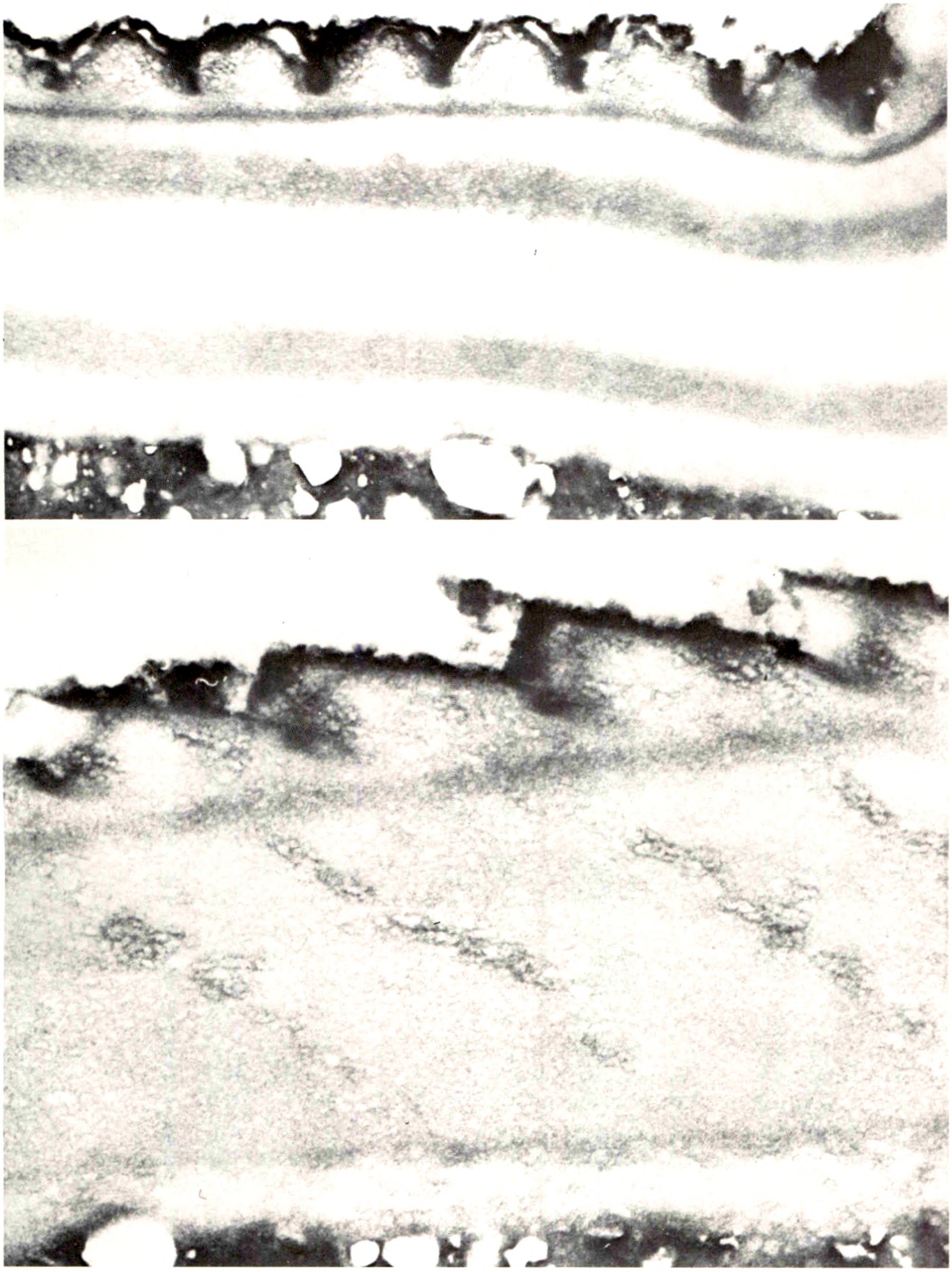

Fig. 15 (Ashton and Cook). Case 4. Top, Worm fragment shows typical nematode cuticle (electron micrograph, ×31,000). Bottom, Another area of the nematode cuticle showing transverse annulations and oblique cuticular tubules (electron micrograph, ×31,000).

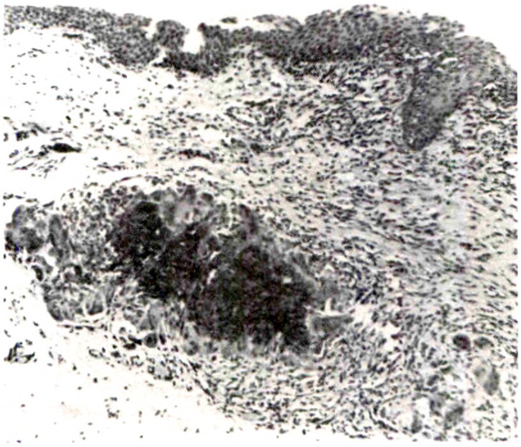

Fig. 16 (Ashton and Cook). Case 5. Section of conjunctiva. A subepithelial nodule consists of a central eosinophilic mass containing nuclear debris surrounded by epithelioid cells and giant cells; numerous eosinophils are present (hematoxylin and eosin, ×112).

by worms) the condition was unilateral; in the remaining four cases (Nos. 5, 6, 9, and 12) the lesions were bilateral and recurrent. The geographic distribution was as follows: Jerusalem, seven; Uganda, five; United Kingdom, nine; Sudan, one; 59% overseas and 41% in the United Kingdom. The 22 cases were collected between 1958 and 1977, but when one recalls that biopsies are not performed in many of these lesions, that they are treated expectantly and may disappear spontaneously, we can be sure that this is a much commoner clinical condition than is implied by our data. No particular seasonal incidence emerged when the dates of onset of symptoms in the group were analyzed. In many cases only limited clinical details were provided and laboratory investigations were inadequate. It seems the ocular condition is essentially local in character, because except for occasional reports of blood eosinophilia, Case 12 was the only one to show direct evidence of systemic involvement.

Reports of similar ocular granulomas—Episcleral or subconjunctival nodules attributed to hypersensitivity, whether of bacterial, toxic, chemical, rheumatoid or unknown cause, have been reported by several authors,[15-21] but although these lesions had necrotic or fibrinoid centers and in some respects a similar ultrastructural pathology,[22] they did not show giant cells or eosinophils as typical features and only superficially resembled the histology of the cases shown here. Bernardino and associates[23] studied the histology of 37 cases of solitary nodular conjunctivitis and described granulomas with giant cells and a variable number of eosinophils, which they believed to be reactions to elastotic fibers in inflamed pingueculae. The quality of their illustrations, however, does not permit comparison with the histology of our own cases although the histologic descriptions are similar. The allergic granulomas described by Harkavy,[24] including one with conjunctival nodules, were predominantly of vascular origin. The histology of the phlyctenular-like nodules described by Aoki, Tamura, and Sugano[25] in Japanese females is similar to that in our own cases and the nodules were of the same nature. The eosinophilic granuloma containing Charcot-Leyden crystals found by Howard[26] in the conjunctiva of a boy with filariasis was also comparable.

Reports of similar nonocular granulomas—Except for the eye, allergic granulomas have been described in various organs as a result of sulfonamide therapy[27-29] (note Figure 10 of Rich[29]), and in Loeffler's syndrome (simple pulmonary eosinophilia) in which the allergic agents reported are extremely diverse, including myocobacterium tuberculosis, helminth infestations (for example, ascaris, taenia, ankylostoma, *Trichuris*), *Endamoeba histolytica*, pollens of various types, drug reactions (to penicillin, aspi-

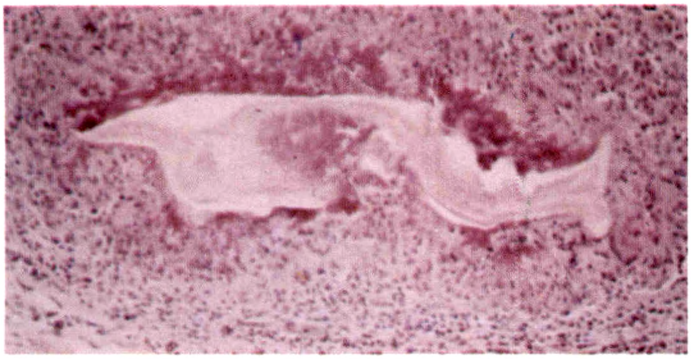

Fig. 2 (Ashton and Cook). Case 1. Another granuloma from this case showing a presumed hyaline worm fragment centrally. Note the surrounding eosinophilic Splendore-Hoeppli precipitate (hematoxylin and eosin, X 224).

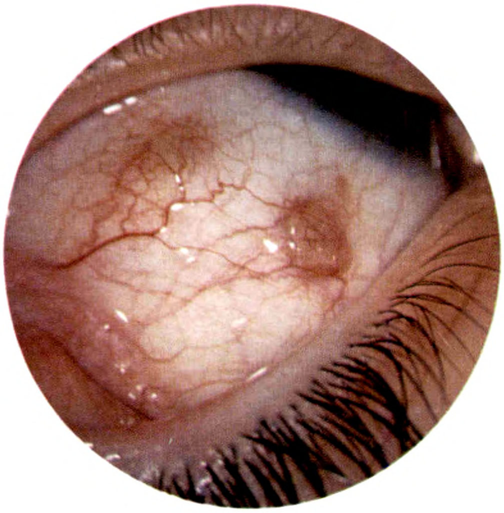

Fig 19 (Ashton and Cook). Case 6. Left eye showing yellow episcleral nodules in superior temporal quadrant with mild hyperemia.

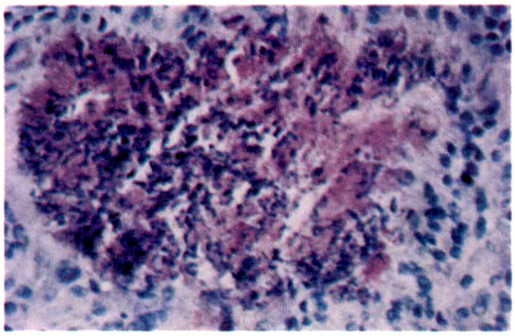

Fig. 20 (Ashton and Cook). Case 6. Section of conjunctiva showing nodule consisting of a granuloma with an intensely eosinophilic center with nuclear debris surrounded by mononuclear cells and a moderate number of eosinophils (hematoxylin and eosin, X 200).

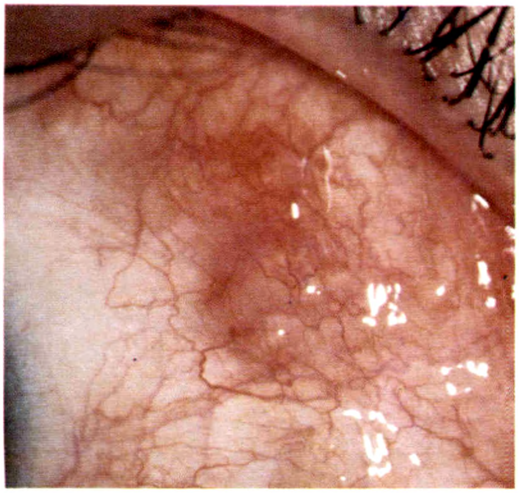

Fig. 21 (Ashton and Cook). Case 12. Right eye. Yellow confluent nodules in the outer bulbar subconjunctival tissue with associated hyperemia.

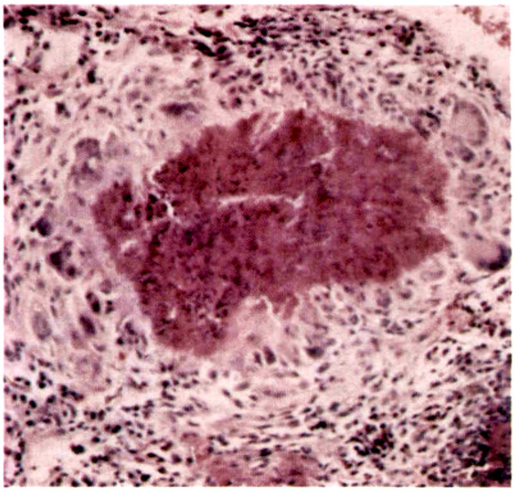

Fig. 22 (Ashton and Cook). Case 12. Section of conjunctiva shows a granuloma with eosinophilic center containing nuclear debris and surrounded by giant cells, epithelioid cells and eosinophils (hematoxylin and eosin, X 280).

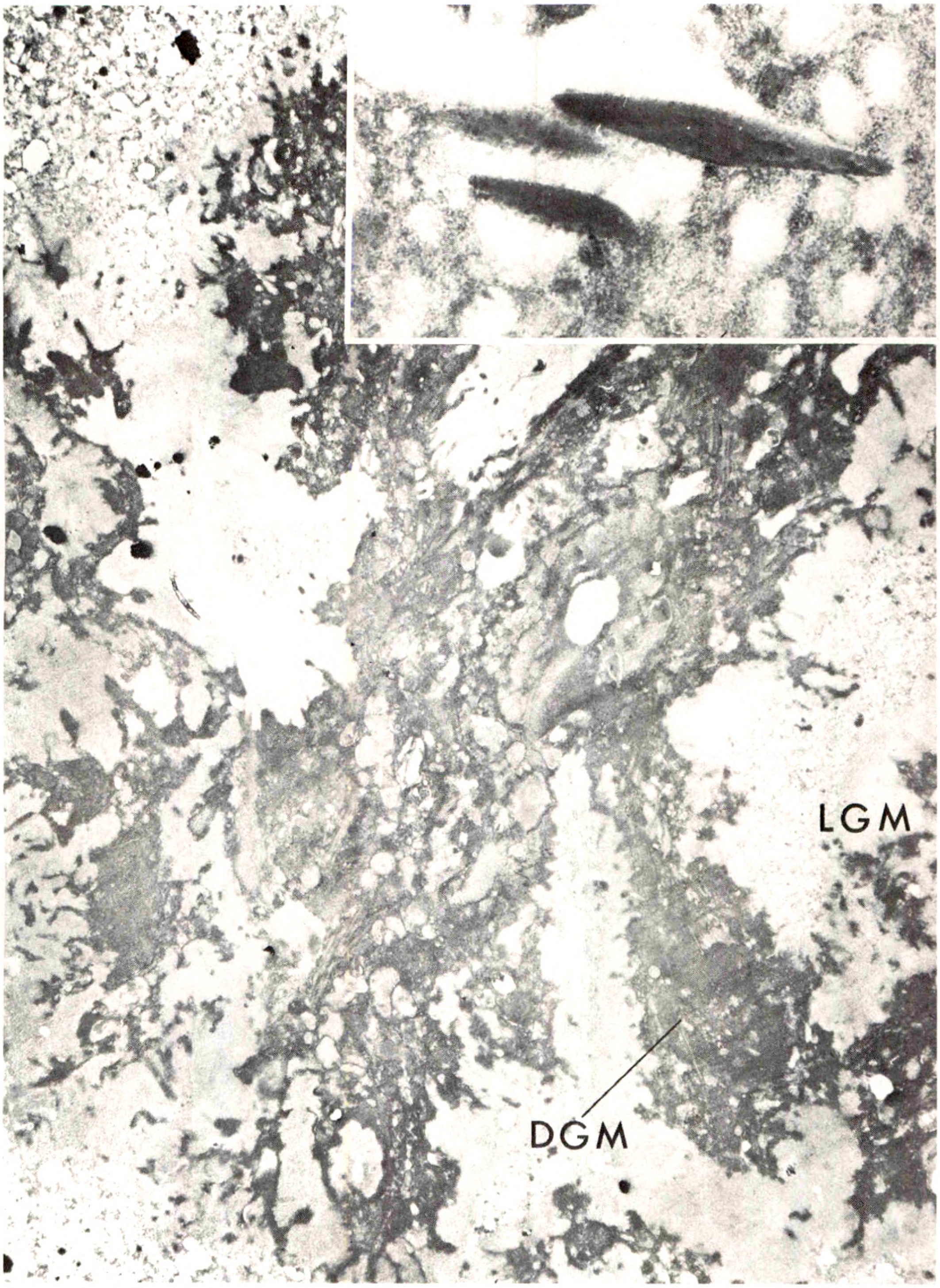

Fig. 17 (Ashton and Cook). Case 5. Nodule shows the characteristic Splendore-Hoeppli precipitate consisting of irregular deposits of electron-dense (DGM) and translucent (LGM) granular material with cellular debris and Charcot-Leyden crystals (inset) (electron micrograph, ×4,800; inset, ×22,100).

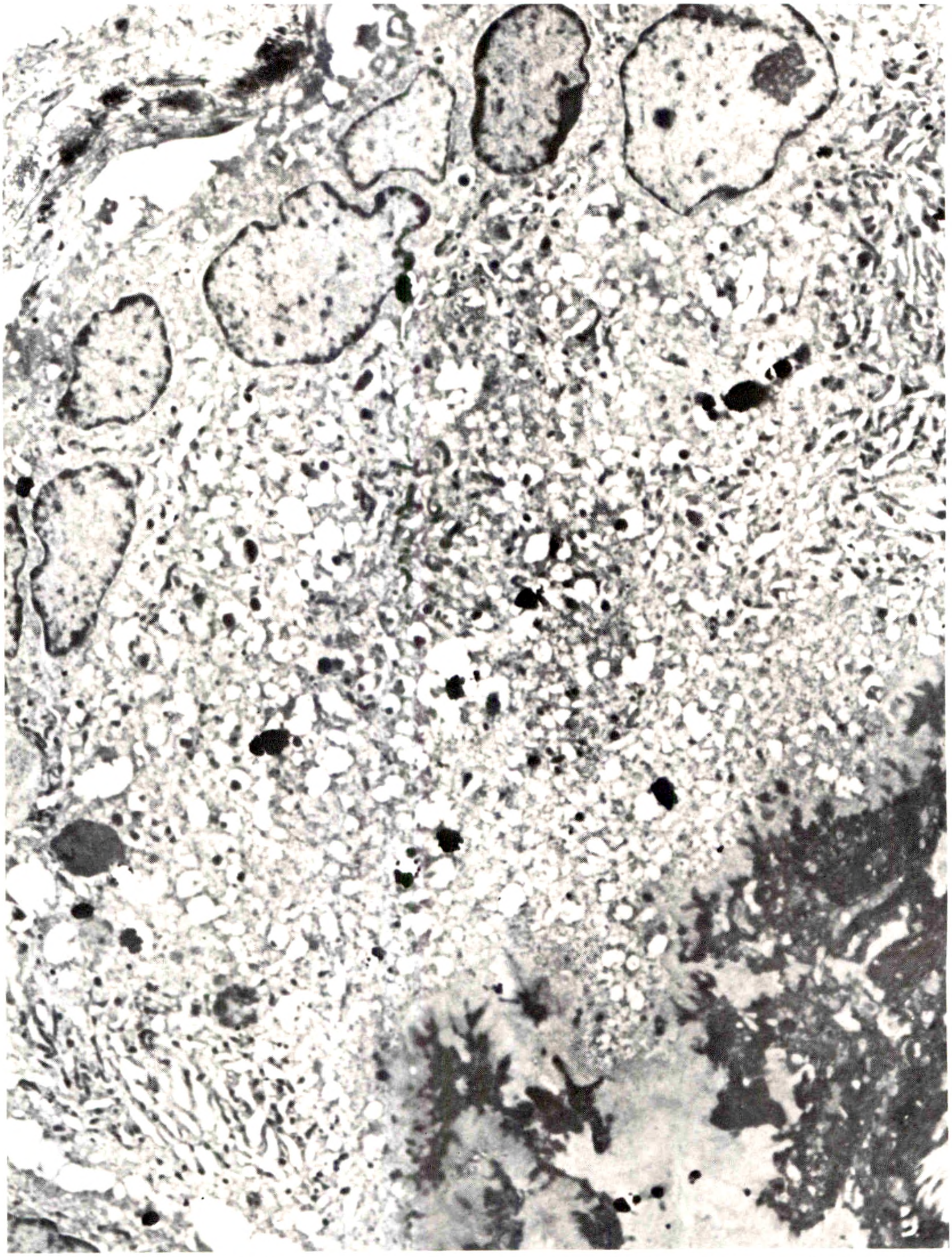

Fig. 18 (Ashton and Cook). Case 5. Another area of specimen shown in Figure 17. A giant cell with multiple nuclei is seen engulfing the Splendore-Hoeppli precipitate (lower right) (electron micrograph, × 4,800).

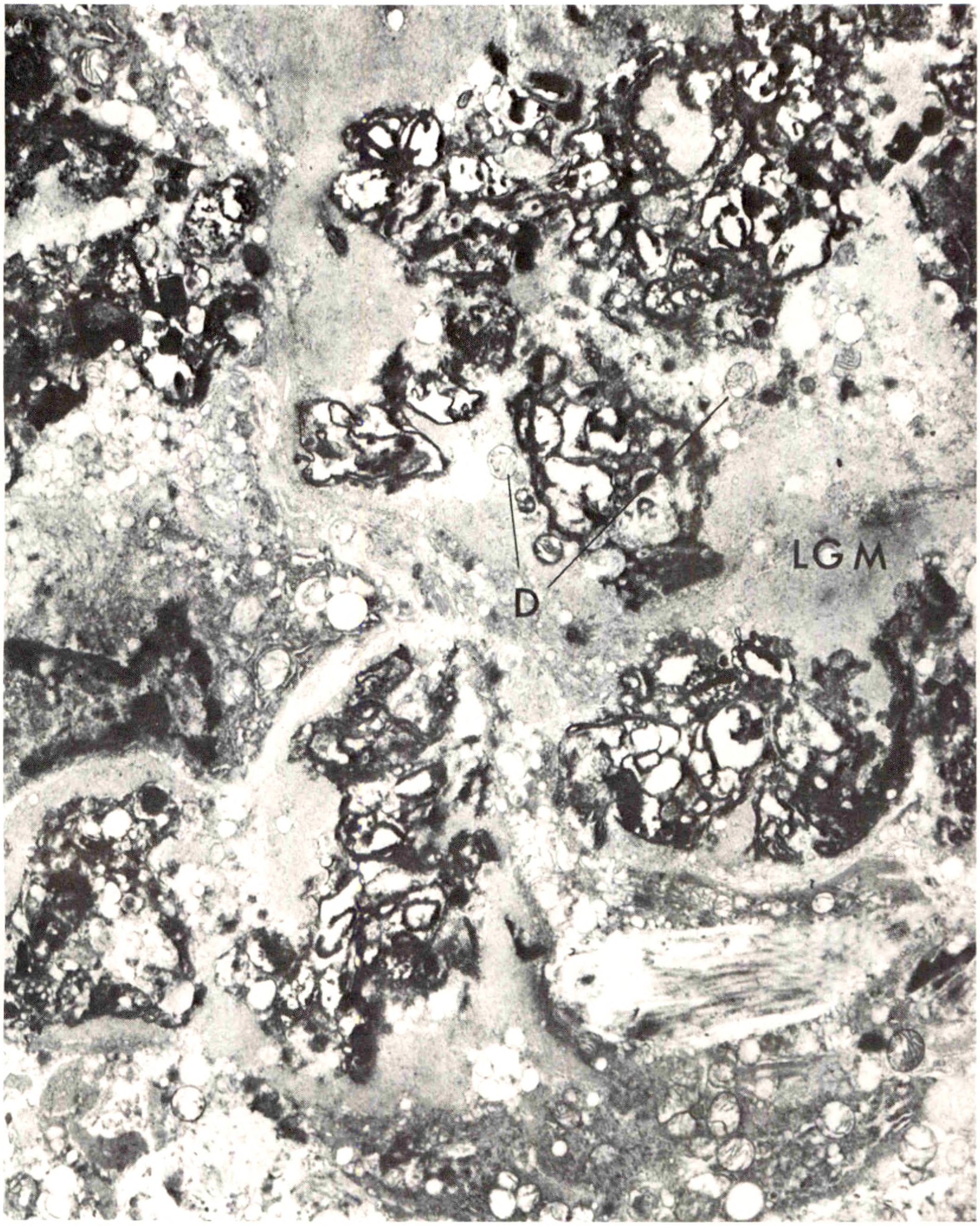

Fig. 23 (Ashton and Cook). Case 12. Typical Splendore-Hoeppli precipitate. Note cellular debris (D) and electron-translucent (LGM) granular material (electron micrograph, ×10,000).

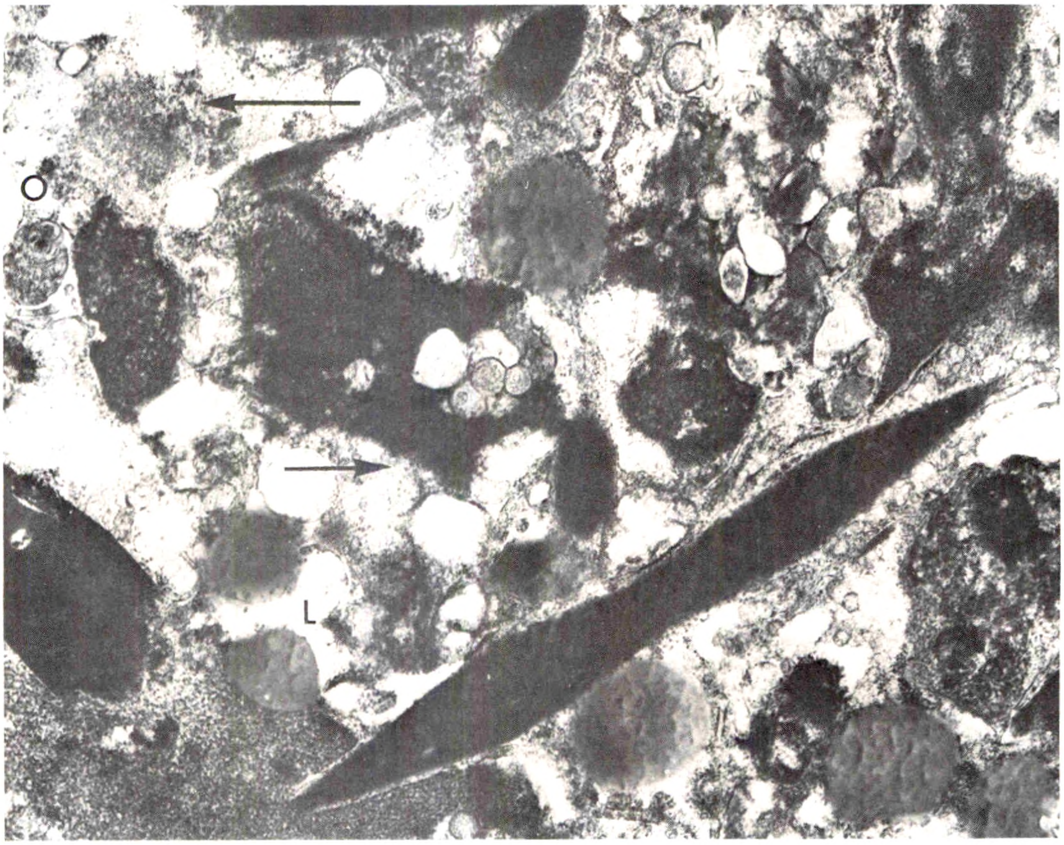

Fig. 24 (Ashton and Cook). Case 12. Granuloma showing acicular Charcot-Leyden crystals, free cytoplasmic organelles (O), lipid globules (L), and granular material (arrows) (electron micrograph, ×30,000).

rin, nitrofurantoin, sulfonamide) and cases of unknown cause.[30] Bronchopulmonary aspergillosis has been reported to be the most common cause of pulmonary eosinophilia among asthmatic patients in Britain.[31] Nodular granulomas including an example in the conjunctiva, have been described in the lungs, liver, kidney, and skin and are regarded as distinct from periarteritis nodosa.[32-34] Histologically, these lesions appear related in some way to our cases but they are not identical. Only one of our cases showed an involvement of collagen fibers in the central core, and fungi were not found.

The exact histologic appearance we are describing was reported by Abel and associates[35] in allergic granulomatosis of the stomach; no causative agent was found, but the response to corticosteroid treatment was dramatic. Similar granulomas have also been described in a case of asthma (Loeffler's syndrome) by Smith,[36] in persistent insect bites of skin,[37] and in children with allergic manifestations.[38] In cases of asthma where the granulomas are extravascular, the clinical course is usually benign[39,40]; this description fits in well with our Case 12.

Allergic granulomas of this kind have also been described throughout the body in visceral larva migrans caused by *Toxocara canis* in a 2-year-old child,[41] and in an infant.[42] They have also been de-

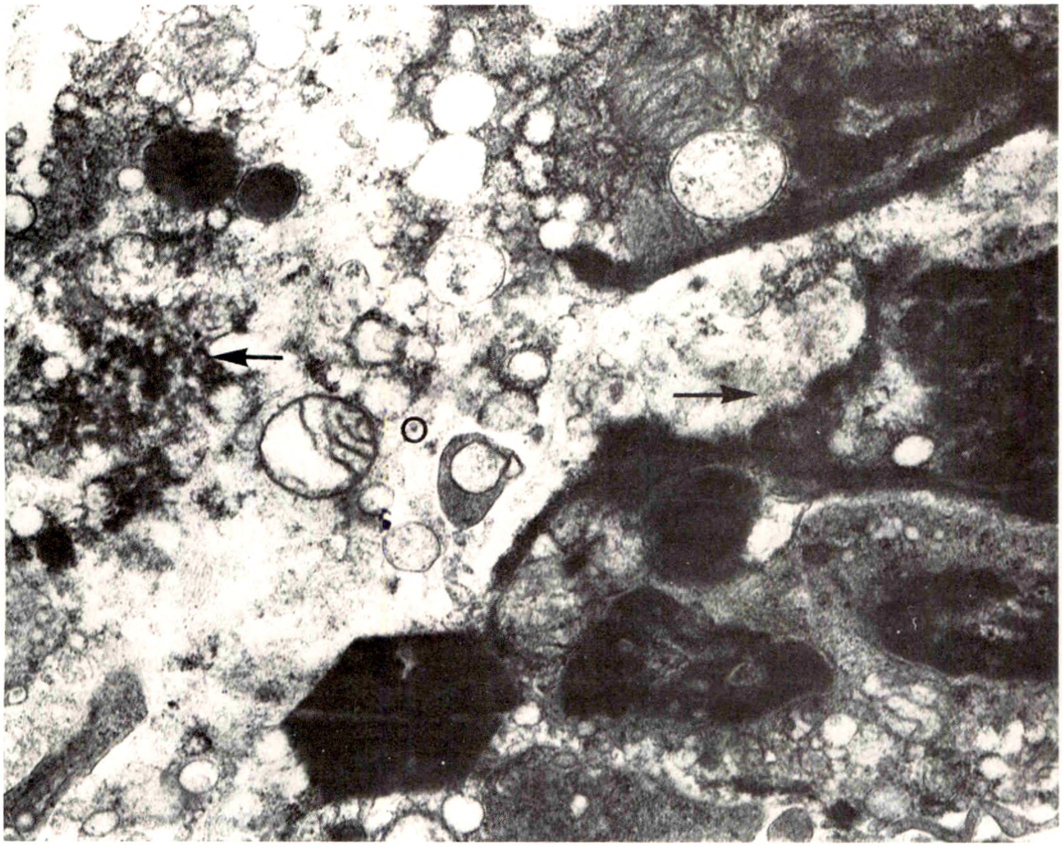

Fig. 25 (Ashton and Cook). Case 12. Another area of Figure 24 showing hexagonal Charcot-Leyden crystals, cytolysis with released organelles (O), and granular deposits (arrows) (electron micrograph, ×39,000).

scribed in the prostate gland associated with asthma,[43-45] and in one such report,[46] were compared with granulomas caused by larval ascariasis as had been described by Mercer and associates[47] (although their case may have been one of toxocariasis). Similarly, a syndrome of young children, disseminated visceral lesions associated with extreme eosinophilia, showed multiple granuloma identical with our ocular cases[38]; in none of the eight cases reported, however, were ascaris larvae or other parasites indicated and the antigen was undetermined. These cases might well have been caused by toxocariasis, now recognized as the commonest cause of visceral larva migrans.[48] However, no trace of a worm remained. This applies also to the granulomas suspected as being caused by *Toxocara* by Heiner and Kevy,[49] and Brain and Allan.[50] *Toxocara* larvae were found in only four of 20 cases of visceral larva migrans described by Snyder[51]; apparently they are quickly destroyed in the tissues.

Nature of the granulomas in the present series—Our cases evidently belong to a group of granulomas that are manifestations of chronic localized immune complex disease, that is, a focal anaphylactic reaction with cellular injury and extracellular release of potent lytic enzymes, leading to necrosis and often to damaged collagen (well shown in the cornea by

Mohos and Wagner[52]), and yet they have a distinct and uniform histology. The lesions are sharply demarcated. They are walled off from the surrounding tissue by a granulomatous giant cell reaction that surrounds a central mass of fibrinoid material that has nuclear debris associated with a dominant infiltrate of eosinophils. Occasionally the giant cells and islands of epithelioid cells seem to have originated from the epithelium. Involvement of collagen is inconstant and vascular involvement is minimal. The appearances correspond to the Splendore-Hoeppli phenomenon, but these special appearances may reflect the presence of a particular type of antigen (the characteristics of which are thought to influence the nature and extent of granulomas[53,54]), or to its slow, persistent, and focal accumulation as may be produced or released by living or dead organisms.

This uniform histologic appearance is exemplified in our 22 cases, and the nematodes found in four cases (all unilateral), may point to a similar etiology. No doubt we should continue to search for such a causative agent in all future cases of this kind. We recall here the reports previously quoted of similar granulomas in visceral larva migrans and emphasize that failure to demonstrate a parasite by no means excludes this etiology since the evidence can be destroyed through rapid disintegration. Moveover, in a sensitized subject granulomas can develop wherever particulate antigen may lodge, so that even emboli of parasitic fragments might initiate a reaction.

Conjunctival nodules and worms—The finding of worms in our four cases, three from Uganda and one from the United Kingdom, was not surprising in those from Uganda, because this exact entity was reported by Owen and Hennessy[55] who described small yellow nodules in the bulbar conjunctiva of 11 Ugandan natives, in three of which unidentified nematodes were found. It occurred to Owen and Hennessy that there was some connection between these bulbar nodules and two other entities common in Uganda, namely, edema of the eyelids (referred to by Elliott[56] as "bung eye" and attributed to insect bites) and proptosis, which the authors called "bulge eye." Bung eye, bulge eye, and conjunctival nodules may be found separately or in combination, and Owen and Hennessy[55] concluded, on the basis of an identical and definite histopathology, that these entities probably had a common cause, a nematode, which differed only in the location of the tissue into which the worm had burrowed. We share this view with regard to our cases from Jerusalem and North Africa.

No worm could be identified by the above authors, and they speculated that a nematode of the genus *Thelazia* might be responsible, because it had previously been reported from China[57-59] and California.[60-63] These were adult worms measuring 8 to 13 mm in length, lying free in the conjunctival sac, where in man they produce only superficial irritation. The only case attributed to the larvae of *Thelazia*, which invaded the lower eyelid, was reported by Howard,[64] but his color illustrations indicate that the parasite he described was more probably *Demodex folliculorum*. We considered a *Demodex* as a possible cause of conjunctival granuloma, but our electron micrographs of this ascarid showed a different structure from that of the nematodes, so that we have made no progress with this idea.

More than 40 years after the report of Owen and Hennessy,[55] Poltera[65] published a histologic study of 40 conjunctival granulomas in Uganda; 18 showed worm fragments, and 22 worm tracks only. The clinical and histologic features were comparable with those in our series and the sizes of the fragments were in the same range. Additionally, Poltera[65] found female worms bearing sheathed microfilariae and he concluded that they belonged to the *Loa* group. The existence of loiasis in Uganda has, however, been denied[66]; conversely, Price[67] considered it to be widespread and published a picture of a conjunctival nodule[68]; Nnochiri[69] claimed to have found the filarial worm *Loa-loa* in an ocular lesion in a Ugandan boy. However, identification of the worms has not been clearly established.

Another nematode, *Habronema*, a parasite of the stomach wall of the horse, which develops in and is carried by various muscid flies, can produce a granulomatous or nodular conjunctivitis in this animal, and a case of bung eye in a baby was attributed to habronemic larvae by Bull.[70] Although Bull described the typical histologic reaction seen in our own cases, the worm was not found, but this was attributed to its having been rapidly destroyed. Habronemic infection in horses is uncommon in the United Kingdom.

Dirofilaria conjunctivae, a filarial nematode of dogs in Europe and Asia has been identified in granulomas excised from human eyelids,[71,72] and in the orbit.[73-75] It has not yet been reported in the United Kingdom; but is widespread throughout warm countries including North Africa, and is much larger than the worm fragments we have found.

Other worms known to involve the eye include *Onchocerca volvulus*, *Ascaris*, *Toxocara*, *Oxyuris vermicularis*, and *Sparganum mansoni*. Only in the last case, however, have nodular conjunctival granulomas been described. The larvae of this cestode are considerably larger than those we have found.

Man is exposed to many worms other than those classed as human parasites, and *Toxocara*, as the commonest cause of visceral larva migrans in temperate climates, needs special consideration. The conjunctiva could act as a portal of entry. In warm and moist conditions the ova readily hatch to release actively invasive larvae, and theoretically, conjunctival contamination with dirt containing ova might explain localized conjunctival lesions without systemic involvement. This possibility does not, however, seem to have been thought worthy of previous consideration.

Although *Trichinella spiralis* infection of the conjunctiva or sclera has not been reported, the parasite cannot be excluded from the list of possible etiologic agents. Trichinellosis has appeared in almost every country in the world and it has been suggested by Kozar[76] that Great Britain is one of those with a relatively serious trichinosis problem. It may be particularly relevant that in mild infections no characteristic clinical signs are visible and the illness may well be attributed to a variety of causes.

Other possibilities are the larvae of flies such as the Warble fly (*Oestridae*) one of which, *Dermatobia hominis*, is frequently found in the skin of man in Central and tropical South America, and has been shown to cause chalazion-like lesions in the eyelids and conjunctiva.[77] We have, however, studied the ultrastructure of *Oestrus ovis* but the cuticle did not show the transverse annulations found in our cases. Portions of insects should also be considered as exciting agents; there are many published examples.[78]

Except for their classification as nematodes, the nature of the worm being considered is still unknown. The cause is even more puzzling when no worm is found, as in our two cases from Uganda, our seven cases from Jerusalem, and one case from the Sudan, but especially in our nine cases from the United Kingdom.

The question arises as to whether a worm may have been the cause in all of these cases, but that it disintegrated by the time of biopsy, leaving no trace even by electron microscopy, or whether we can go no further than the histologic diagnosis of allergic granuloma, implying an immune complex reaction where the initiating antigen may be of widely differing kinds. It is highly probable that all our cases from Jerusalem, Uganda, and the Sudan have a similar parasitic etiology. With our positive findings in one case from the United Kingdom, that would account for 14 of our 22 cases (64%).

Our review of reported nonocular cases with identical or similar etiology indicates that uniformity of histologic findings does not necessarily imply a common etiopathogenesis. In our Case 12, for instance, the presence of recurrent bilateral conjunctival nodules, nonocular nodules, tissue eosinophilia in the temporal muscle, and the presence of asthma, all point to a systemic condition. Our failure to show any causative agent despite exhaustive clinical and laboratory investigation, leads to the conclusion that this case corresponds more closely to the type of allergic granulomas occurring with atopic allergy associated with asthma as described previously. In this group of cases, however, the responsible allergen, whether organismal or otherwise, has often not been identified, and in view of our own negative findings in conjunctival nodules where the cause is almost certainly parasitic, they also cannot be entirely dismissed as in a different etiologic category.

This study does not resolve the problem of the cause in those cases where no worm was found, and although circumstantial evidence suggests that a similar infestation is likely to explain a higher proportion of these cases, others with identical histology remain unexplained. Future cases should be more completely investigated with special attention directed to a parasitic etiology, with the provision of tissue for microdissection, immune-labeling techniques, and electron microscopy, because it is possible that the solution, or solutions, to this conjunctival puzzle may elucidate the wider problem of nonocular allergic granulomas.

SUMMARY

We studied the clinical and pathologic features of 22 cases of granulomas of the conjunctiva or eyelids. All cases showed the histologic features of the Splendore-Hoeppli phenomenon, that is, a giant cell and eosinophil granulomatous reaction to an antigen-antibody precipitate originally described in relation to parasites or fungi. In four of seven typical cases selected for detailed description unidentified nematodes were found to be the cause of the condition. In light of these findings together with a review of similar "allergic granulomas" reported both in ocular tissues and elsewhere in the body, we considered the cause in the remaining cases. All 22 cases may have been caused by nematodes, as seems, probable in 14 of them, or the causative antigens may have been of widely different kinds. Although in our cases all ocular granulomas had an identical histology, this study did not resolve the problem of those cases where no causative agent was found.

Thorough investigation of such cases in the future and the demonstration of their cause may elucidate the wider problem of nonocular allergic granulomas.

ACKNOWLEDGMENTS

The following provided us with the pathologic material and clinical details: Professors Barrie Jones, J. Pepys, and E. S. Perkins; Messrs. M. A. Bedford, R. Blach, A. J. Boase, H. F. T. MacFetridge, C. G. F. Munton, Redmond Smith; and Drs. K. L. Batten, K. G. Hadija, A. Knudsen, and A. Ali Eltayed. The following provided case reports: Dr. R. Barry (Case 3), Dr. A. Garner (Cases 8 and 18), Dr. J. Harry (Cases 5, 7, 11, 12, and 16), Dr. G. Morgan (Case 10), and Dr. K. Valteris (Case 6).

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PRIMARY MUCINOUS SWEAT GLAND CARCINOMA OF THE EYELID SIMULATING METASTATIC CARCINOMA

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Rosen, Kim, and Yermakor[1], and Grizzard, Torozyski, and Edwards[2] recently reported examples of an unusual variant of eccrine sweat gland carcinoma of the eyelid. Most sweat gland carcinomas pose no diagnostic problem histologically; however, this variant is often confused with inflammatory conditions, benign tumors, and metastatic eyelid carcinomas.[1,2] Because it is difficult to diagnose correctly, we report herein the clinicopathologic and ultrastructural details of such a tumor.

CASE REPORT

In October 1975, a 78-year-old man with a five-month history of blepharoptosis and a two-month history of a painless, palpable mass on his upper right eyelid was referred to us for examination, and was subsequently admitted to University Hospitals of Cleveland in February 1976. Physical examination revealed a diffuse, insensitive, firm, nontranslucent mass over the entire upper right eyelid and most of the lower right eyelid through the lateral canthal area (Fig. 1). There was no eyelid or eyelid margin ulceration or lash loss. This mass was fixed to the skin but not to the orbital rim or periostium. The palpebral fissure was narrowed. Eyelid mobility was moderately reduced. Visual acuity was 6/6 (20/20) in both eyes; pupils, extraocular muscles, conjunctiva, anterior segment, and fundi were normal. No proptosis could be measured. Results of examination of preauricular, submandibular, and cervical lymph nodes were normal. A complete physical examination was unremarkable except that the barium enema indicated diverticulosis and malposition of the sigmoid colon.

A full-thickness upper right eyelid block resection was performed for diagnosis but no therapy was begun. He was further observed as an outpatient. Examination 14 months later revealed an increase in the size of the upper right eyelid mass with increased extension into the lower right eyelid. When seen 26 months after initial diagnosis, the mass in his right eyelids had enlarged slightly.

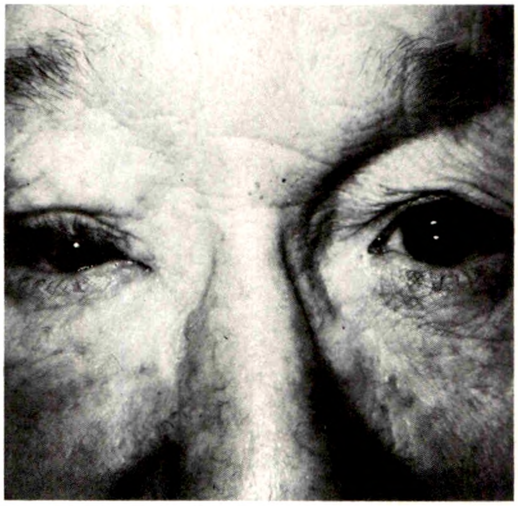

Fig. 1 (Thomas, Fu, and Levine). Diffuse thickening and induration of the right upper and lower eyelids. Compare with uninvolved left eyelid.

Under microscopic examination, the overlying skin of the eyelid was normal. However, there were abundant lymphocytes in the perivascular spaces of the upper dermis (Fig. 2). Some of the lymphocytes were mixed with atypical cells, which appeared in isolated form or as loose aggregates (Fig. 2). In the deeper dermis, atypical cells appeared in compact clusters traversing the collagen and skeletal muscle fibers (Fig. 3). A pattern of atypical cells arranged in a single row was also evident in focal areas (Fig. 3). The nuclei of the atypical cells were usually round to oval, but some were irregular and hyperchromatic. Nucleoli were found occasionally. Mitotic figures were rare. The cytoplasm was abundant, vacuolated, and foamy. Some cells contained a single discrete vacuole with a signet-ring appearance (Fig. 3).

Because of the vacuolated appearance of the cytoplasm, a mucicarmine stain was done which indicated mucicarminophilic material in the cytoplasm, and a PAS stain also revealed diastase-resistant material in the cytoplasm. We diagnosed adenocarcinoma.

We further examined the lesion by electron microscopy, for which a small portion of the tissue was fixed in 2.5% phosphate-buffered glutaraldehyde, postfixed in 1% osmic tetroxide for one hour, dehy-

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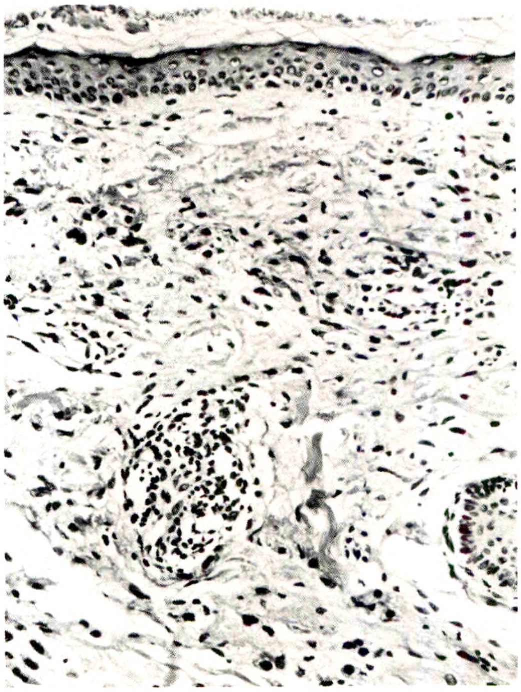

Fig. 2 (Thomas, Fu, and Levine). Low-power photomicrograph showing perivascular lymphocytic infiltration and scattered abnormal cells in the dermis, suggesting inflammation (hematoxylin and eosin, ×200).

drated in a graded series of ethanol solutions, and embedded in Spurr medium. Thin sections were cut with a diamond knife, stained with uranyl acetate and lead citrate, and examined with an electron microscope.

Electron micrographs showed that the tumor cells contained numerous, irregularly shaped and sized, membrane-bound vacuoles. Some were filled with fibrillar or loose flocculent secretory material, whereas others appeared empty (Figs. 4 and 5). Golgi membranes and mitochondria were abundant. Bundles of tonofilaments were interspersed between the organelles. The cell membranes often showed microvillous interdigitation with the neighboring cells that were joined by desmosomes. Intracytoplasmic, as well as intercellular, lumens were frequently found (Figs. 4 and 5). The electron microscopic study clearly indicated the glandular nature of the tumor cells.

DISCUSSION

Two other reports described the same clinical and pathologic findings.[1,2] Initial diagnosis in both of these reports was granular cell myoblastoma. The diagnoses of the consulting pathologists included granular cell myoblastoma, inflammatory granulation tissue, xanthoma, histiocytoma, a proliferative mast cell lesion, neurogenic tumors, nevi, and metastatic carcinomas, possibly of breast ori-

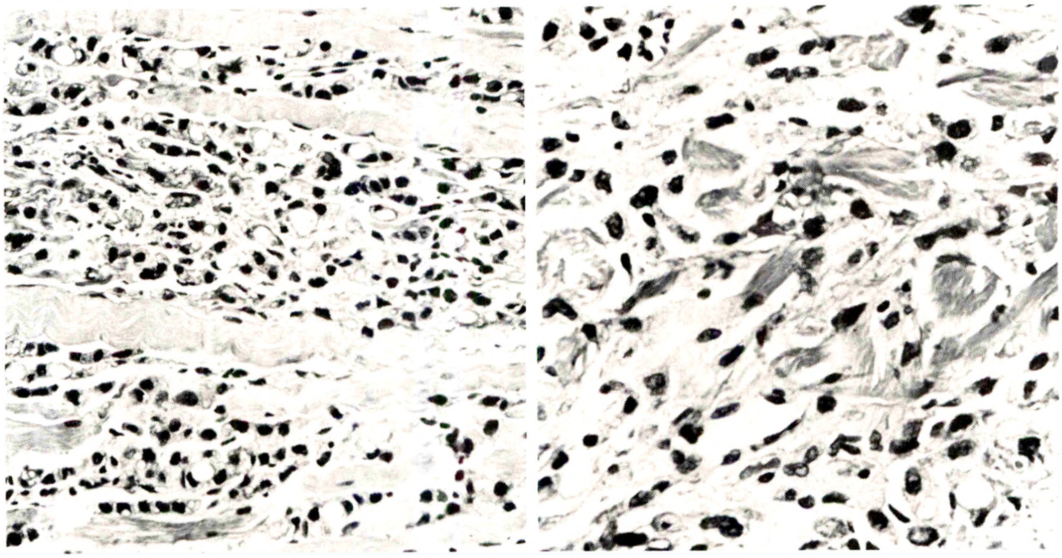

Fig. 3 (Thomas, Fu, and Levine). Left, Clusters of tumor cells infiltrate collagen fibers. Many tumor cells contain discrete clear vacuoles and simulate signet-ring cells. In the lower portion, atypical cells are arranged in a single-row pattern. Right, Some of the tumor cells demonstrate nuclear hyperchromasia and irregularity. Notice vacuolated, foamy cytoplasm (hematoxylin and eosin; left, ×225; right, ×285).

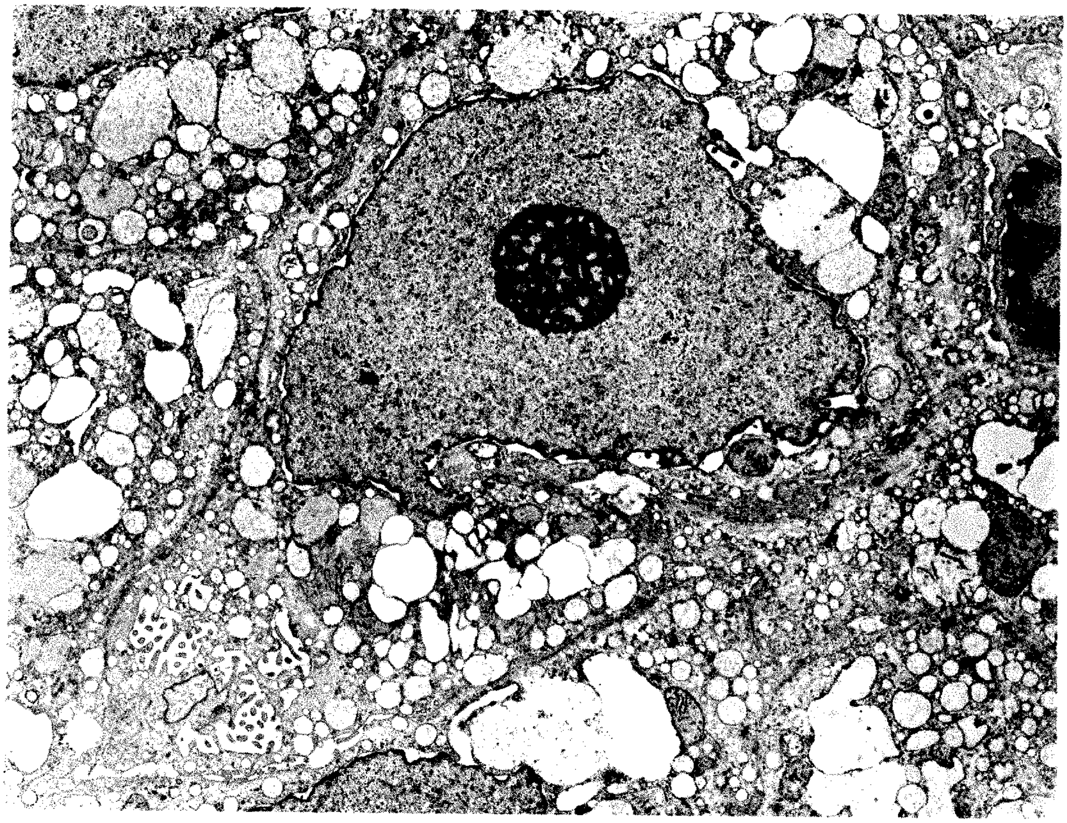

Fig. 4 (Thomas, Fu, and Levine). The tumor cells contain numerous vacuoles of different sizes. Some are filled with fluffy, fibrillar material; others are empty. An intercellular lumen is present at the left lower corner. A prominent nucleolus is also present (×7,200).

gin. These lesions were finally diagnosed as eccrine sweat gland carcinomas by special stains and electron microscopic studies, complete clinical examinations, and follow-up information.[1,2]

The appearance of granular and eosinophilic cytoplasm and relatively uniform nuclei suggests a diagnosis of granular cell myoblastoma, and the foamy, vacuolated appearance of some of the tumor cells suggests a histiocytic or xanthomatous tumor. Intermingling of tumor cells with inflammatory cells (usually lymphocytes) also suggests a diagnosis of inflammatory condition. However, the pattern of dermal infiltration and nuclear irregularity indicate a neoplastic process. Special stains (PAS and mucicarmine) make a mucin-producing adenocarcinoma apparent.

The histologic appearance of our case as well as two other reported cases[1,2] is indistinguishable from that of histiocytoid carcinoma of the eyelid secondary to carcinoma of the breast, an entity first reported by Hood, Font, and Zimmerman.[3] In eight of their 13 female patients with metastatic carcinoma of the eyelid originating from the breast, the tumor cells were histiocytoid. These lesions were frequently diagnosed as benign conditions such as granular cell myoblastoma, xanthelasma, histiocytoma, chronic inflammation, blastomycosis, and granulation tissue. In five of these 13 cases, metastasis to the eyelid preceded recogni-

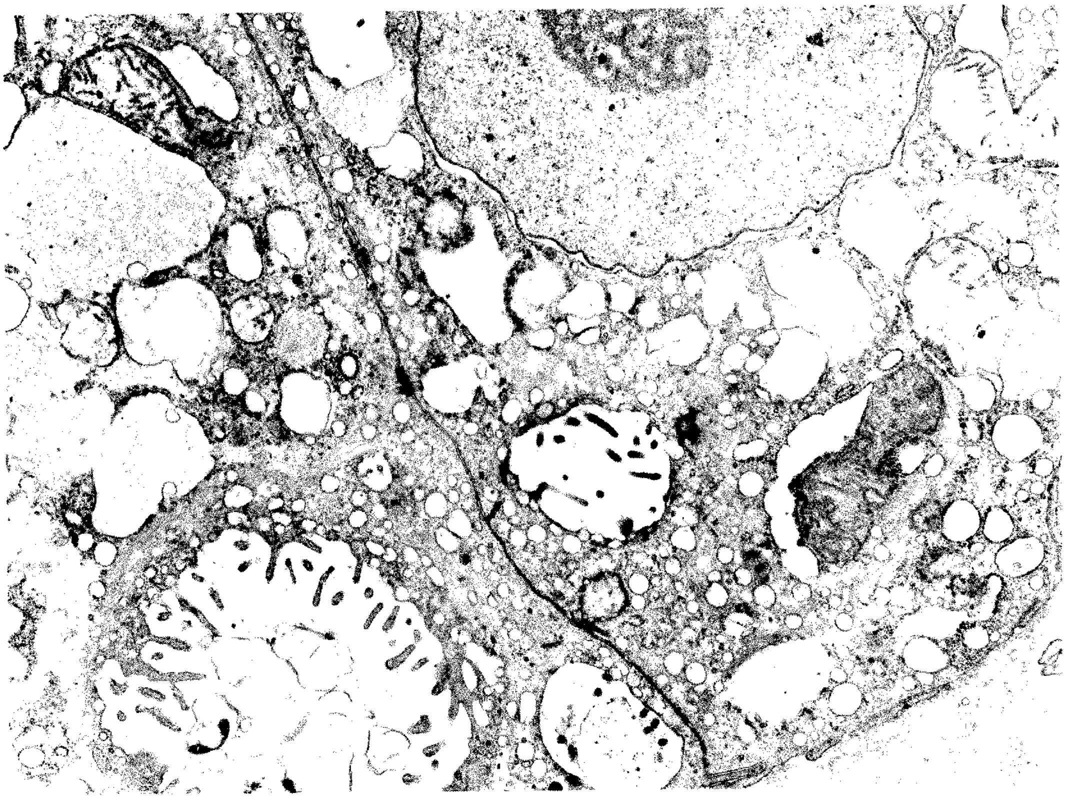

Fig. 5 (Thomas, Fu, and Levine). Two tumor cells joined by a desmosome. There are three intracytoplasmic lumens with microvilli projection into them. Notice multiple secretory vacuoles (×11,200).

tion of primary breast carcinoma. Therefore, a correct histologic diagnosis of these lesions is essential as it may indicate cancer elsewhere in the patient's body.

Our patient and two other patients [1,2] previously described were men. No primary cancer was found during complete clinical examination of various organ systems, including careful examination of the breast. In this case, absence of finding a primary tumor elsewhere in the man's body over a 26-month period suggested that the eyelid tumor was primary.

Electron microscopic findings for this tumor confirmed a malignant glandular neoplasm. The granular eosinophilic cytoplasm seen by light microscopy corresponds to the presence of secretory granules observed by electron microscopy. The foamy appearance is related to multiple vacuoles and intracytoplasmic lumens seen under the electron microscope. These findings closely resemble those in the normal eccrine gland and its carcinoma as previously described by Rosen, Kim, and Yermakor.[1] The presence of intracytoplasmic lumens and abundant tonofilaments in this tumor also simulates carcinoma of the mammary glands, especially the lobular infiltrating variant, as described by Battifora,[4] Fisher,[5] and Steinbrecher and Silverberg.[6] Certain similarities exist between neoplasms of sweat gland and mammary gland origin, because normal sweat glands and mammary glands have a similar cellular component.[5] The light and electron microscopic features of this eyelid lesion are indistinguishable from those of lobular infiltrating and signet-ring carcinomas of the breast.[6]

Stout and Cooley,[7] and Berg and McDivitt[8] reported that the histopathology of sweat gland carcinoma may vary considerably. Because most sweat gland carcinomas contain glandular or papillary formation, they pose no diagnostic problem. However, the anaplastic, poorly differentiated category, and the variant, such as in the present case, have not been recognized as a distinct entity because of the relative infrequency of these tumors.

The neoplasm we report here seems to have a long clinical course characterized by multiple recurrences. In the patient described by Rosen, Kim, and Yermakor,[1] the tumor recurred but had not metastasized over a five-year period. Grizzard, Torozyski, and Edwards[2] reported that in their patient the tumor recurred eight years after initial incision; and metastasis occurred in the preauricular lymph nodes ten years after initial incision. Following radiation therapy, there was no evidence of recurrence for 18 months. Our patient's lesion has persisted over the last 26 months and has not metastasized.

Adenocarcinoma of the eyelid, especially with a histiocytoid appearance, should alert the pathologist to the possibility of metastatic carcinoma from the breast in the female patient. If the male patient has adenocarcinoma of the eyelid, he should be examined completely to rule out a neoplasm elsewhere. If the results of the examination are negative, the possibility of eccrine sweat gland carcinoma of the eyelid should be considered, and the diagnosis confirmed by electron microscopy.

SUMMARY

A 78-year-old man suffered an unusual variant of sweat gland carcinoma of the eyelid. This disorder is seldom recognized, and is frequently confused with inflammation, benign neoplasms, and metastatic carcinomas. On the basis of clinicopathologic and ultrastructural features found by both light and electron microscopy, we diagnosed sweat gland carcinoma of the eyelid. We considered the possibility of metastasis from a primary carcinoma elsewhere in the patient.

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CONGENITAL RETINO-PIGMENT EPITHELIAL MALFORMATION, PREVIOUSLY DESCRIBED AS HAMARTOMA

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In 1973 Gass[1] described five patients with unusual fundus lesions, which he interpreted on clinical grounds as hamartoma of the pigment epithelium and retina. McLean[2] later published a report of another case. We describe herein the clinical and angiographic findings in six more patients. In one patient, for the first time, we were able to obtain a histologic study that revealed a peculiar malformation of the retina and pigment epithelium.

CASE REPORTS

Case 1—In 1962, a 16-year-old boy was referred for consultation because of a "choroidal hemangioma" in the right eye that had been observed for about three years and recently had shown clinical evidence of growth. Visual acuity in that eye had been reduced for an unknown period of time. The general health of the patient was good, and his family history was noncontributory for eye disease.

On initial examination, his visual acuity was R.E.: 6/60 (5/50); L.E.: 6/4.8 (5/4). Motility was within normal limits, and he had no strabismus. Slit-lamp examination of both eyes revealed no abnormalities. On ophthalmoscopic examination, all abnormalities were confined to the posterior fundus of the right eye: surrounding the optic disk, there was a slightly elevated lesion of about 8 × 10 disk diameters that was grossly irregular in its outline with feathery margins. Its most impressive features were a greenish-blue pigmentation and a marked tortuosity of both veins and arteries that was confined to the area of the lesion. The more peripheral vessels appeared to be straight and stretched, but otherwise normal. Preretinal membranes, which could have explained the tortuosity, were seen neither clinically nor on the fundus photographs. The retina had lost its transparency and obscured the underlying choroidal pattern. Much of the lesion, nasally to the disk as well as in the central macular area, showed a peculiar dark greenish-blue pigmentation, which seemed to be deep under the retina at the level of the pigment epithelium and gradually blended into the normal background pigmentation. Where abnormal retina blended into normal tissue, intraretinal cystic spaces were seen. A zone of reflexes demarcated the area of pathology from the normal fundus (Fig. 1).

During the following months, several observers thought the lesion was definitely growing. Therefore, five months after the initial visit, the eye was enucleated because of a suspected melanoma. Histology revealed hyperpigmentation and, presumably, hypertrophy of the retinal pigment epithelium combined with malformation and thickening of the overlying sensory retina (Figs. 2 and 3).

Case 2—A 6-year-old boy was referred for consultation because of an amblyopia in the right eye and a "macular pucker" in the left eye. His mother had noted a squint for about one year. The boy's general health was good, and his medical and family history were unremarkable.

On initial examination, an uncorrected hyperopia was found, and his visual acuity was R.E.: +2.75 sph 0.1; L.E.: +5.25 sph = −1.5 cyl. A 30° 1/25. There was a minimal right exotropia of about 5 degrees, and fixation was eccentric in both eyes. Slit-lamp examination revealed no abnormalities. Ophthalmoscopically, the right eye was normal except for a "little traction" above the macula that was not further described or documented. In the macula of the left eye was a greenish-blue pigmentation of about 1 × 2 disk diameters located at the level of the pigment epithelium and gradually blending into the normal fundus pigmentation. The retina directly overlying the pigmentation was thrown into horizontal folds, and the yellow pigment was obvious. There was considerable distortion and traction of the major temporal vessels toward the 2 o'clock meridian. The surrounding retina, in an area of about 4 × 6 disk diameters, appeared slightly

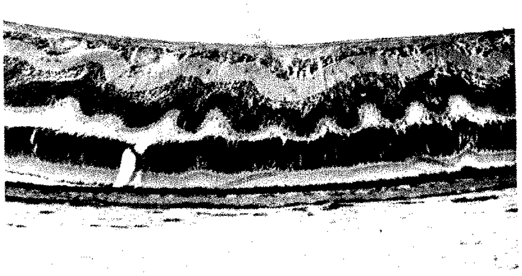

Fig. 2 (Laqua and Wessing). Case 1. Photomicrograph of a horizontal section through the area nasal to the disk. Note the disturbance of retinal layering in the absence of preretinal membranes and the thickening of the nuclear layers (hematoxylin and eosin, × 125).

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AMERICAN JOURNAL OF OPHTHALMOLOGY 87:34–42, 1979

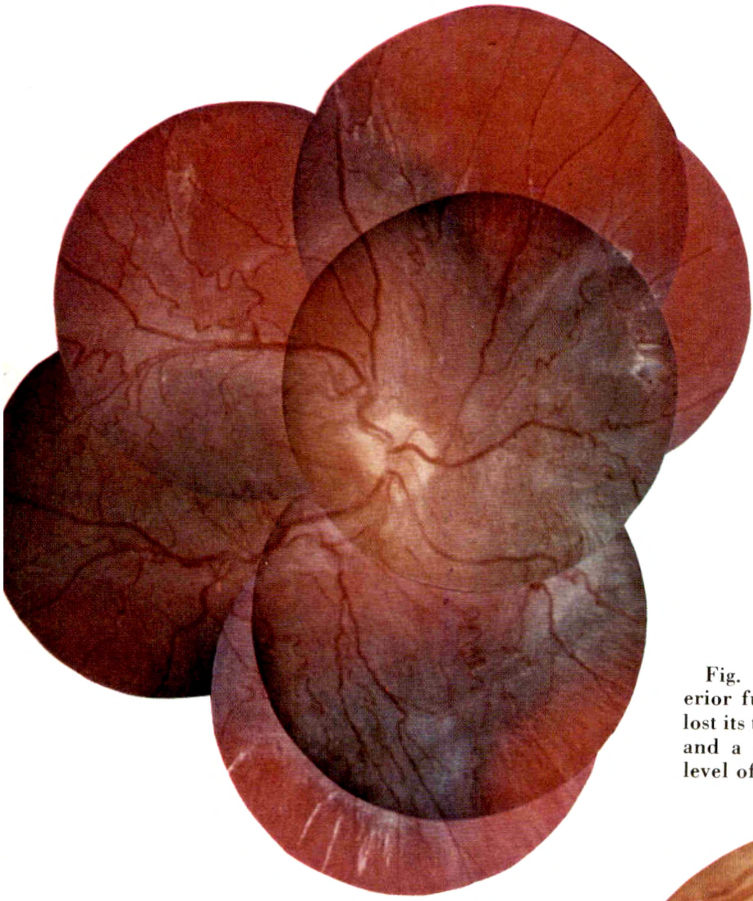

Fig. 1 (Laqua and Wessing). Case 1. At the posterior fundus the retina appears thickened and has lost its transparency. There is tortuosity of all vessels and a peculiar greenish-blue pigmentation at the level of the pigment epithelium.

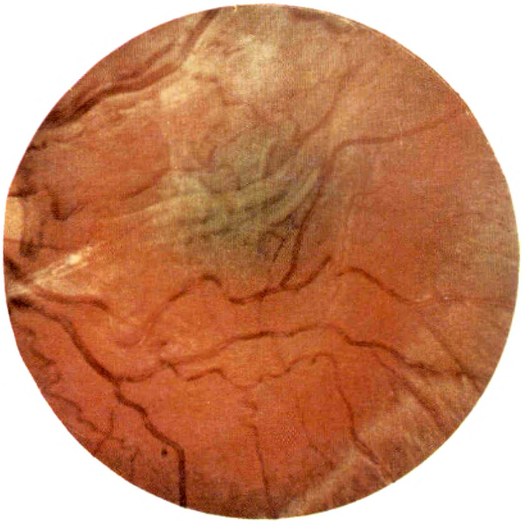

Fig. 4 (Laqua and Wessing). Case 2. In the macular area there is considerable distortion of the vessels, and the retina overlying the greenish-blue pigmentation is thrown into horizontal folds. A few preretinal membranes are seen along the superior arcade. Reflexes demarcate the area of disease.

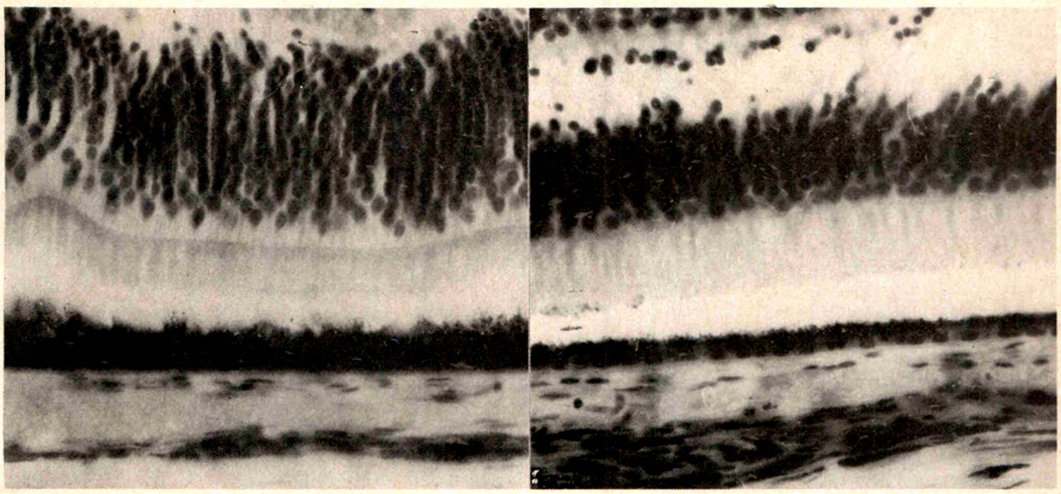

Fig. 3 (Laqua and Wessing). Case 1. Composite photomicrograph of a horizontal section comparing pigment epithelium cells from nasal side (left) with cells located more temporally (right). Where a greenish-blue pigmentation was noticed clinically, the pigment epithelial cells appear hyperpigmented and hypertrophied (hematoxylin and eosin, × 315).

grayish-white and less transparent, but was not obviously thickened. Thin, gray preretinal membranes were present, especially along the superotemporal arcade. A ring of reflexes demarcated the area of pathology from the normal fundus (Fig. 4). Outside the lesion, the retina and pigment epithelium were unremarkable. The vessels were straight and stretched, but otherwise normal. Spectacles were prescribed and vision improved from 6/60 (20/200) to 6/15 (20/50) in the right eye, but remained 6/150 (20/500) in the left eye. During a follow-up period of one year the lesion did not change.

Case 3—A 3-year-old boy was referred because of an intermittent esotropia and a "macular hemorrhage" in the left eye. Esotropia had been noted since age 3 months. His general health was good, and the family history was noncontributory for ocular disease.

On initial examination, vision was R.E.: 6/6 (20/20); L.E.: 6/30 (20/100). Motility was within normal limits, and he had no strabismus. He had an uncorrected hyperopia of +2.0 diopters in each eye. Slit-lamp examination of both eyes revealed no abnormalities. Ophthalmoscopically, all abnormalities were confined to the posterior fundus of the left eye. In the macular area, there was a greenish-blue pigmentation of about 1 × 2 disk diameters, which was deep under the retina and blended imperceptibly into the normal fundus pigmentation. The overlying retina, in an area of about 4 × 5 disk diameters, was slightly thickened, had lost much of its transparency, and appeared grayish-white. There was tortuosity of all vessels with traction toward the pigmented center of the lesion. Some of the larger vessels seemed to disappear for short distances, but no preretinal membranes were visible. A ring of reflexes demarcated the area of involvement from the normal fundus. Peripherally to the lesion, the vessels were straight and stretched. The fundus periphery was otherwise normal (Fig. 5). During a follow-up period of six months, the lesion did not change.

Case 4—A 4-year-old boy was referred by a

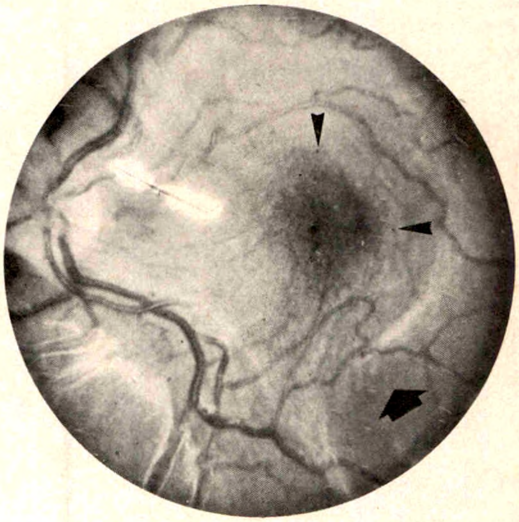

Fig. 5 (Laqua and Wessing). Case 3. Note the central area of pigmentation (small arrows), outline of thickened retina (large arrow), and vessel distortion. Reflexes demarcate area of pathology from normal fundus.

neurologist with a tentative diagnosis of a "congenital retinal fold" in both eyes. At 4 months of age, the boy had suffered from a pachymeningitis haemorrhagica and subsequently had developed mild symptoms of a cerebral palsy. On initial examination, the child was not cooperative and visual acuity could not be ascertained. Motility seemed to be within normal limits, and he had no strabismus. There was an uncorrected hyperopia in both eyes of +5.0 sph. Slit-lamp examination revealed a normal anterior segment in both eyes. Ophthalmoscopically, the posterior pole was distinctly abnormal in both eyes. In the macular area of the right eye there was a greenish-blue pigmentation of about 1.5 disk diameters that was located at the level of the pigment epithelium and imperceptibly blended with the normal color of the fundus. The overlying retina in an area of about 4 × 6 disk diameters revealed tortuosity of the vessels with some traction toward the pigmented center. Although the vessels were obscured from view for short distances, no preretinal membranes were discernible. Ophthalmoscopically, there was no measurable elevation of the retina, but the retina appeared less transparent and partly obscured the choroidal pattern. The optic disk was eccentrically inside the area of pathology, but appeared normal (Fig. 6).

Similar, but more marked changes were observed at the posterior pole of the left eye. The area of involvement was about 5 × 7 disk diameters, included the optic disk nasally, and again was demarcated against the normal fundus by a ring of reflexes. There was a similar greenish-blue pigmentation deep under the retina, and the retina appeared less transparent. The greenish-blue pigmentation and the retinal involvement roughly matched in size, and both were most prominent temporally where dense grayish-white preretinal membranes were seen. Tortuosity of the vessels was also most pronounced temporally, and there was marked traction of the larger vessels toward the 3 o'clock meridian, which resulted in a fan-like distortion of the vascular pattern and extended from the disk area toward the periphery. Directly overlying the fovea, the retina was thrown into coarse horizontal folds, and the yellow pigment was obvious (Fig. 7).

During a three-year follow-up period, the fundus lesions did not change. When the child was 7 years old, his visual acuity was R.E.: 6/30 (20/100); L.E.: 6/60 (20/200).

Case 5—When drafted into the army, a 20-year-old man was found to have poor vision in his right eye. A "pigmented tumor" was seen in the macula, and the patient was referred for further diagnostic studies. On initial examination in 1958, his visual acuity was R.E.: hand movement at 2 feet; L.E.: +0.5 sph = +1.0 cyl A 90° 1.2. Motility was within normal limits, and he had no strabismus. Slit-lamp examination of both eyes and ophthalmoscopy of the left eye revealed no abnormalities. All pathology was confined to the posterior pole of the right eye.

At the posterior pole and extending slightly inferotemporally, there was a slightly elevated fundus lesion of about 3 × 5 disk diameters that was well demarcated against the normal fundus by a ring of reflexes. The optic disk was inside the lesion at its nasal margins. The retina appeared thickened and grayish-white and obscured the choroidal pattern. The retinal vessels were markedly tortuous, in contrast to the straight and stretched vessels outside the lesion. At the surface of the lesion, a fine filigree pattern could be recognized and probably represented abnormal retinal capillaries. The vessels were sheathed. Preretinal membranes were present, but not marked. At the inferior margins of the lesion, where whitish and thickened retina blended into normal retina, a dark pigmentation was visible. It seemed to be deep under the retina, probably at the level of the pigment epithelium. The pigmentation extended beyond the area of retinal thickening, but remained inside the ring of reflexes. The overall color of the lesion was a peculiar greenish-blue, which seemed to be the result of an interference of

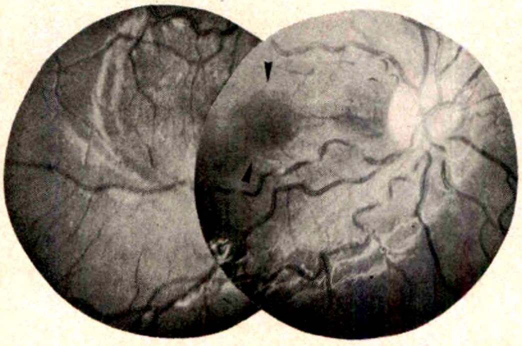

Fig. 6 (Laqua and Wessing). Case 4 (right eye). Arrows outline the pigmentation. Vessels are obscured from view for short distances, but no preretinal membranes are recognizable.

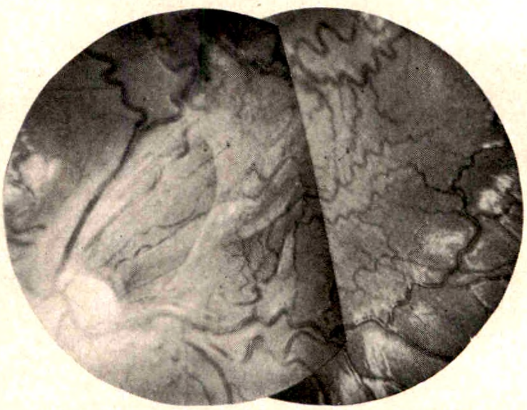

Fig. 7 (Laqua and Wessing). Case 4 (left eye). Preretinal membrane is seen overlying the central macular area. Area of pigmentation and area of retinal involvement roughly match in size.

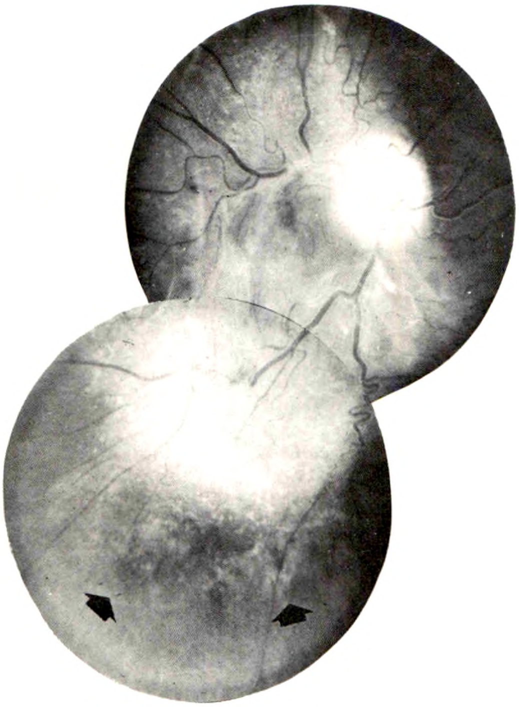

Fig. 8 (Laqua and Wessing). Case 5. Pigmentation (arrows) extends beyond area of thickened, whitish retina.

the dark pigmentation of the outer portion with the grayish-white color of the inner portion of the lesion (Fig. 8).

Intraretinal cystic spaces appeared where abnormal retina blended into normal retina. A rim of subretinal fluid was evident at the superotemporal margins.

Comparison of the color fundus photographs taken in 1958 and those taken at an examination in 1977 revealed only minimal changes. Size, elevation, and the overall appearance of the lesion remained unchanged. Only one larger vein had changed its course over a short distance. The smaller vessels, however, could not be recognized as easily as before, either because of increasing retinal thickening or preretinal membrane formation. The filigree pattern had become more prominent, and the formerly homogenous pigmentation at the inferior margins later appeared granular.

Angiography performed in 1974 and 1977 revealed blockage of the background choroidal fluorescence, which corresponded to the area of pigmentation. The major retinal vessels were angiographically unremarkable. The most striking finding, however, was a peculiar network of abnormal capillaries in the superficial retina, which leaked dye in the late phases. These capillaries probably corresponded to the fine filigree intraretinal pattern visible ophthalmoscopically (Fig. 9).

Case 6—A 10-year-old boy was referred to us because of a "melanoma" in his left eye. Since age 1 year, the parents had noticed that the child did not see well with his left eye and that there was some exodeviation. Except for a hearing problem of unknown cause and recurrent bronchitis, his medical history was unremarkable. The family history was noncontributory for ocular disease.

On initial examination, his visual acuity was R.E.: -0.5 sph 1.0; L.E.: +4.75 sph 0.1. There was a left exotropia of about 12 degrees. The anterior segments of both eyes were within normal limits. Ophthalmoscopically, the right eye was normal, but the posterior pole of the left eye was distinctly abnormal: a grayish-blue lesion was seen that was about 4 × 6 disk diameters. The bluish hue was most marked above the fovea and apparently was caused by pigmentation under the retina. The retina appeared slightly thickened and gray and obscured the choroidal pattern. All vessels were tortuous, and some seemed to disappear for short distances deep into the retinal tissue. Fine preretinal membranes were present, but there was an obvious discrepancy between the marked tortuosity and the amount of

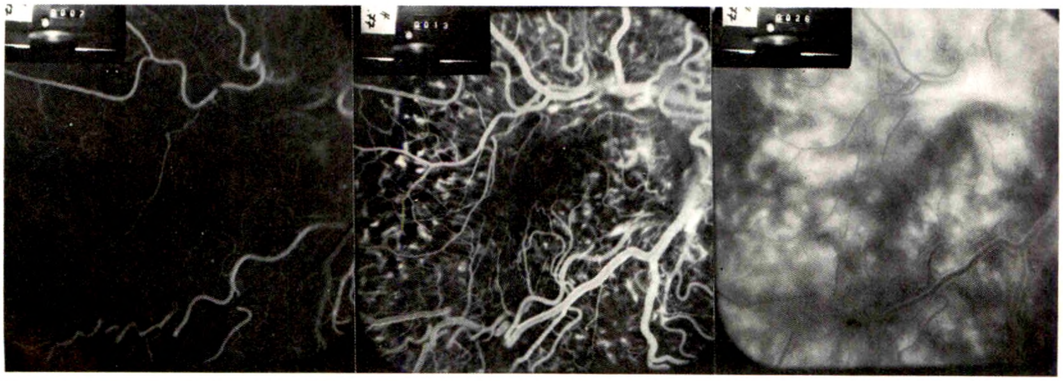

Fig. 9 (Laqua and Wessing). Angiography of Case 5. Note blockage of background choroidal fluorescence in the early phase and network of abnormal, leaking capillaries.

preretinal membrane formation. Superotemporally from the fovea where the retinal thickening was most marked, some subretinal fluid was detected. A ring of fine, radially oriented reflexes demarcated the area of pathology from the normal fundus (Fig. 10).

Angiographically, there was blockage of the choroidal background fluorescence in an area roughly corresponding to the overall size of the lesion. However, the area of blockage was larger than would be expected from the ophthalmoscopically visible pigmentation. Many abnormal capillaries leaked dye in the late phases (Fig. 11).

Over a four-year follow-up period, the lesion did not change and visual acuity remained L.E.: 6/60 (0.1).

DISCUSSION

The fundus lesions described in our cases and in those reported by Gass[1] and McLean[2] had the following clinical features in common: (1) Each was a slightly elevated greenish-blue or gray lesion in the macular area or fundus periphery and was composed of a flat, outer pigmented portion and a prominent, unpigmented inner portion. (2) The outer pigmented portion consisted of a flat sheet of highly pigmented tissue that gradually blended into the surrounding normal pigment epithelium. (3) The inner portion was composed of thickened retina that had lost its transparency, showed variable degrees of whitening, and obscured the choroidal pattern and much of the outer pigmented portion. (4) The retinal vessels in the areas involved were markedly tortuous, and there was displacement of retinal tissue toward the center of the lesion. Outside the lesion the vessels were straight and stretched. When the lesion was located in the periphery, there often was marked traction of the major vessels extending from the disk toward the peripheral lesion. (5) Tortuosity of the retinal vessels was observed in every case, whereas preretinal membranes were not. There often was a discrepancy between size and thickness of the membrane and the extent of the tortuosity. (6) Angiography was performed in five of 13 lesions. Blockage of the background choroidal fluorescence was seen in three patients and appeared to be present in the remaining two as well, but was not specifically mentioned. The major retinal vessels were tortuous, but otherwise normal. In four cases there was a network of abnormal, dilated capillaries. Leakage in the late phases was evident in three of the

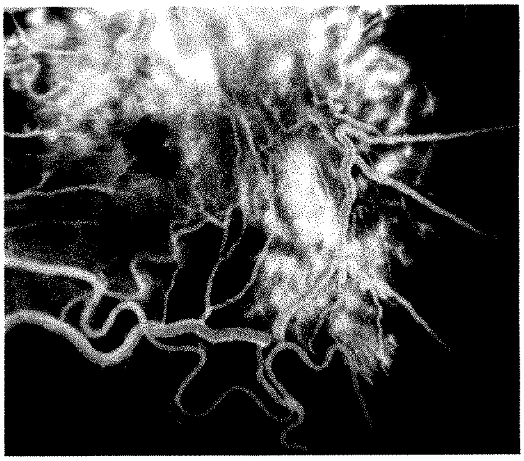

Fig. 11 (Laqua and Wessing). Angiography of Case 6. Note abnormal leaking capillaries and blockage of the background choroidal fluorescence.

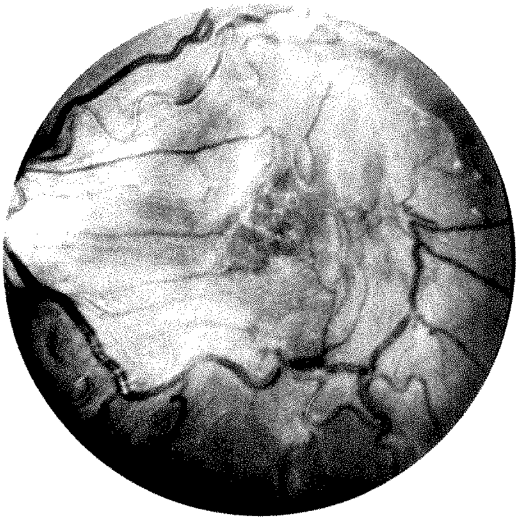

Fig. 10 (Laqua and Wessing). Case 6. Obvious discrepancy between marked tortuosity of inferotemporal vein and the amount of preretinal membrane formation. The tortuosity probably is caused by an intraretinal disturbance of the retinal layering.

four cases. (7) Typically, the lesion was found in otherwise healthy children or young adults who had monocular strabismus, reduced visual acuity, and often hyperopia. Ten of 12 patients were males, and except for in one patient, the lesions were unilateral. Follow-up examinations of up to 19 years did not reveal any significant changes in the size or appearance of the lesions. Frequently, misdiagnosis of a choroidal melanoma or macular pucker occurred in these patients.

One of our cases could be studied histopathologically, which greatly helped to interpret the clinical observations: the outer pigmented portion consists of a layer of hyperpigmented, presumably hypertrophied pigment epithelium and is responsible for the blockage of the background choroidal fluorescence. The inner unpigmented portion consists of thickened, malformed retina that shows a peculiar disturbance of its layering. This intraretinal disturbance of the retinal layering seems to be the main reason for the tortuous retinal vessels. Preretinal membranes are probably only an additional, but less important factor. Secondary changes in the retina such as leakage from abnormal capillaries with cystoid degeneration, sheathing of abnormal small vessels, intraretinal gliosis, and surface contracture caused by preretinal membranes seem to modify the overall clinical picture and result in variable degrees of retinal thickening, distortion, and lack of transparency. When the retinal thickening is less marked, the outer dark-pigmented portion will be the prominent clinical feature and is clearly distinguished from the inner portion (Cases 1 to 4). With increasing lack of transparency and increasing retinal whitening, however, the lesion reveals a diffuse greenish-blue color, and the outer and inner portion may no longer be distinguishable (Cases 5 and 6).

The clinical characteristics of all 13 cases and the histological observation made in one case support the view that this is a benign lesion, probably without any growth potential, and that it is not acquired, but represents a congenital malformation involving the retinal pigment epithelium as well as the overlying sensory retina. Secondary changes in the sensory retina apparently do occur during life, but are not marked. Although the lesion may clinically simulate a tumor, it does not histopathologically impress us as a true tumor or hamartoma.

Gass[1] and McLean[2] thought that the malformation described herein and the juxtapapillary tumors that are called juxtapapillary proliferation of the pigment epithelium,[3,4] hyperplasia of the pigment epithelium,[5,6] and juxtapapillary hamartoma of the pigment epithelium and retina,[7,8] represent variants of the same tumor. The histological and clinical characteristics, however, are sufficiently different to classify them as two different lesions. The juxtapapillary tumors are characterized by extensive gliosis and disorganization of the retina with loss of the regular retinal layering. The pigment epithelium extends anteriorly into the retina and optic nerve head portion of the tumor. Furthermore, the tumor is restricted to the optic nerve head and its immediate vicinity, and the tissues involved show signs of proliferative activity. In contrast, in the malformation described here, the retina remains highly organized, there is no pigment epithelium extension into the retina (even when the optic disk and peripapillary retina are located inside the area of pathology), the lesion may involve a much larger area, and there are no signs of proliferative activity.

The pigmented component of the lesion described herein seems histologically similar to congenital hypertrophy of the retinal pigment epithelium.[9] The changes in the overlying sensory retina, however, are different. In congenital hypertrophy of the retinal pigment epithelium, the photoreceptor cells overlying the area of abnormal retinal pigment epithelium degenerate progressively during life and the retina becomes thinned, whereas in the malformation described herein, the photoreceptor cells are remarkably well preserved and the sensory retina is thickened and malformed. This suggests that either the retinal pigment epithelium in both lesions is functionally different or that the susceptibility of the overlying retinal structures is different.

The clinical features of the malformation in our cases are so characteristic that in a typical case the diagnosis is easy. When the outer pigmented portion is the most prominent clinical feature, the differential diagnosis includes a choroidal nevus, choroidal melanoma, congenital pigment epithelial hypertrophy, and reactive postinflammatory or posttraumatic pigment epithelial lesions. When the inner retinal component is more prominent, one must also consider macular pucker, postinflammatory retinal lesions, retrolental fibroplasia, and the various forms of disciform lesions secondary to choroidal neovascularization. Angiography can be helpful in establishing the correct diagnosis.

Although long-term follow-up did not reveal evidence of growth in any of the reported cases, it recently has been documented that a patch of hypertrophied, hyperpigmented pigment epithelium may enlarge.[10] Therefore, at least the pigmented portion of the tumor might show some growth. This together with increasing surface contracture (as documented in our Case 5) might well have been the basis for the clinical impression of growth in Case 1, which led to enucleation in 1962 before this entity was recognized as a benign lesion.

SUMMARY

Six patients with a congenital malformation of the retina and pigment epithelium had a slightly elevated greenish-blue or gray lesion in the macular area or fundus periphery composed of a flat, outer pigmented portion consisting of a layer of hyperpigmented, presumably hypertrophied retinal pigment epithelium and a prominent, unpigmented inner portion consisting of malformed thickened retina. Marked tortuosity of the retinal vessels was explained by an intraretinal disturbance of the retinal layering and the presence of preretinal membranes. Angiography revealed blockage of the background fluorescence caused by the abnormal retinal pigment epithelium and leakage from dilated, abnormal capillaries. Typically, the lesion was unilateral and was found in children or young adults who had monocular strabismus, reduced visual acuity, and often hyperopia. Most of the patients were males, and their cases were frequently misdiagnosed as tumors.

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THE DIAGNOSIS AND TREATMENT OF SECONDARY GLAUCOMA AFTER HYPHEMA IN SICKLE CELL PATIENTS

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Severely increased intraocular pressure (IOP) was found in four patients with sickle cell hemoglobinopathy after accumulation of small amounts of blood in the anterior chamber. The erythrocytes may have become sickled in the aqueous humor, thereby making them rigid and less likely to escape from the eye through ordinary outflow channels. A logjam of sickled erythrocytes in these channels could produce an increase of IOP similar to that occurring in phacolytic glaucoma, hemolytic glaucoma, and ghost cell glaucoma. We attempted to determine whether or not autologous erythrocytes do actually become sickled in the anterior chambers of sickle cell patients and whether surgical removal of the erythrocytes has a beneficial effect on the secondary glaucoma. Details of the four patients studied are found in the Table and in Figures 1 through 8.

DISCUSSION

Autologous erythrocytes can become sickled in the anterior chamber of the eye. All patients in this series had a higher percentage of cells sickled in their aqueous humor than in their circulating venous blood (Fig. 8).

The sickle cell hemoglobin C (SC) genotype and possibly the chronic nature of the hyphema (more than eight days) in Case 1 explain the higher percentage of cells that were sickled in this patient's anterior chamber, as compared to those of the patients with sickle cell trait (AS). Conversely, a benign genotype (Hgb AS) and brief incubation do not necessarily prevent sickling from occurring in the anterior chamber of eyes with hyphemas. All three patients with sickle cell trait showed more sickling of erythrocytes in their aqueous humor than in their venous blood. After eight hours in the anterior chamber of the patient who had perforating trauma (Case 4), 18.6% of the erythrocytes were sickled, although only 0.3% of the venous erythrocytes were sickled (Table).

The relatively isolated, stagnant environment within the anterior chamber unfavorably influenced the behavior of erythrocytes in sickle cell trait, which is a condition ordinarily characterized by lack of sickling and absence of pathologic consequences of sickling. In both patients with sickle cell trait and blunt trauma (Cases 2 and 3), the increase in IOP was so severe (Figs. 3 and 5) that early surgical treatment was necessary to protect the vascular perfusion in the patients' optic nerves.

The origin of increased IOP in the patients described was multifactorial. In Case 1, rubeosis iridis was the predominant influence, because paracentesis gave only temporary relief. In Cases 2 and 3, blunt trauma damaged the outflow channels and contributed to the initial increase in IOP. However, the dramatic

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This is an abridgment of a thesis submitted by Dr. Goldberg as partial fulfillment of requirements for associate membership in the American Ophthalmological Society, May 1978.

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TABLE

HYPHEMA PATIENTS UNDERGOING ANTERIOR CHAMBER ASPIRATIONS*

	Case 1	Case 2	Case 3	Case 4
Age (yrs), Race, Sex	46, B, M	11, B, M	15, B, M	40, B, F
Hgb A, %	0	61	57	55
Hgb S, %	53	39	43	45
Hgb C, %	47	0	0	0
Hgb type	SC	AS	AS	AS
Hematocrit, %	40.4	40.1	37.3	38.6
Hemoglobin, g/100 ml	13.8	14.0	12.8	14.3
MCV, cu μ	89	72	78	103
MCH, pg	30.3	25.2	26.7	33.4
MCHC, %	34.1	35.0	34.6	32.4
Duration of hyphema	>8 days	24 hrs	24–48 hrs	8 hrs
Cells sickled, %				
In venous blood	30.7	1.0	0	0.3
In anterior chamber	63.0	9.4	20.0	18.6

*Normal values: hemoglobin (Hgb) A, 100%; Hgb S, 0; Hgb C, 0; hematocrit, 42 to 52% in males and 37 to 47% in females; total Hgb 14 to 18 g/100 ml in males and 12 to 16 g/100 ml in females; mean corpuscular volume (MCV) 84 to 99 cu μ; mean corpuscular hemoglobin (MCH), 27 to 3 pg; and mean corpuscular hemoglobin concentration (MCHC), 32 to 36%.

response to removal of most of the erythrocytes (Figs. 3 and 5) suggests that trabecular blockade was the most important factor in increasing IOP.

Other causes of erythrocyte-induced glaucoma,[1] such as erythrocytic debris and macrophage accumulation (hemolytic glaucoma), iron deposition (hemosiderotic glaucoma), or pupillary block (collar-button clot involving both posterior and anterior chambers), were excluded by the short-term course (one to two days), the appearance of the smeared blood, and the small volume of the anterior chamber blood clots (1.5 to 2.0 mm hyphemas). Although morphologic studies (unpublished observations) do show macrophages with ingested red blood cells only one to two days after anterior chamber injections of erythrocytes, their number is small as compared to the large numbers of red blood cells in the trabecular meshwork.

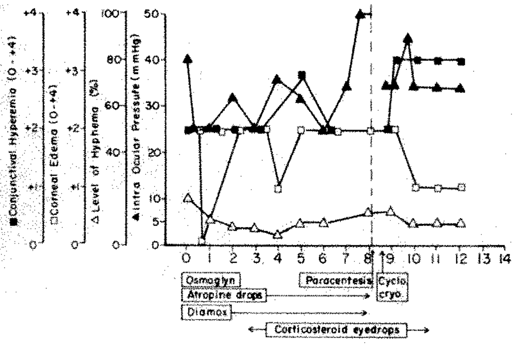

Fig. 1 (Goldberg). Case 1. Clinical course. Note inability to control intraocular pressure (solid triangles) with glycerin (Osmoglyn) and acetazolamide (Diamox).

Trabecular blockage can be created by unsickled erythrocytes, sickled erythrocytes, or by degenerated forms (ghosts) of either variety of red blood cells. Although precise quantitation of these four types of erythrocytes in the trabecular meshwork was impossible, ghosts of either sickled or nonsickled cells constituted a minority of the total erythrocyte burden during the first one to two days after the entry of erythrocytes into the anterior chamber. Rabbit eyes injected with human erythrocytes capable of sickling showed more prolonged hyphemas and more severe in-

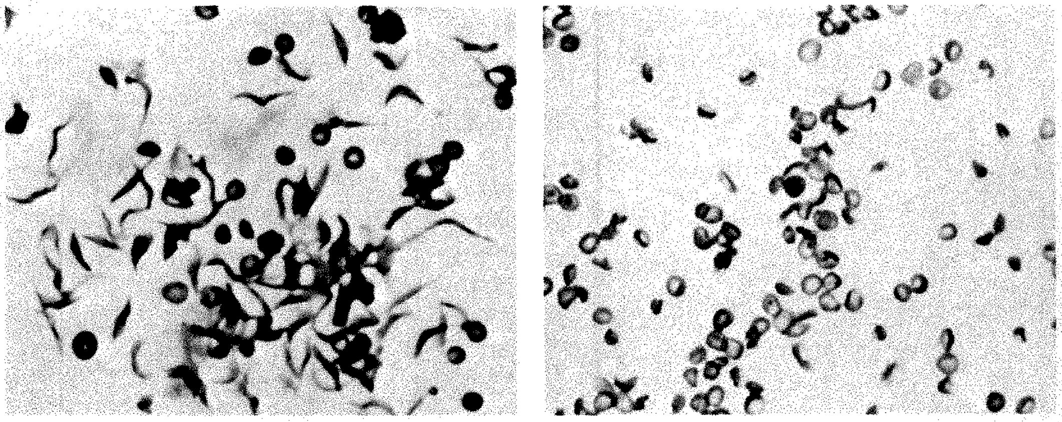

Fig. 2 (Goldberg). Case 1. Left, Hyphema cells show more sickling than in venous blood from same patient. Right, Venous blood shows less sickling than in hyphema cells from same patient.

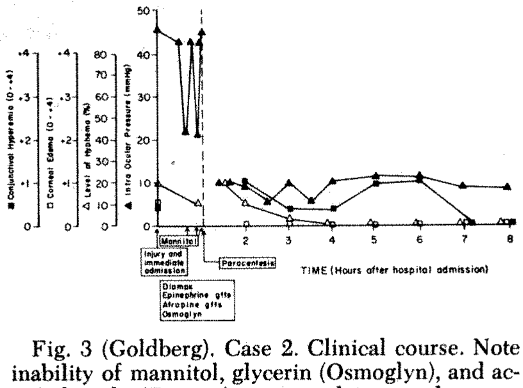

Fig. 3 (Goldberg). Case 2. Clinical course. Note inability of mannitol, glycerin (Osmoglyn), and acetazolamide (Diamox) to control intraocular pressure. Following paracentesis, pressure was normal (solid triangles).

creases in IOP than did rabbit eyes injected with human erythrocytes incapable of sickling (unpublished observations).

In normal, nonsickling individuals, secondary glaucoma caused by bleeding into the anterior chamber can induce optic atrophy by reducing perfusion in that tissue's small blood vessels. Isolated case reports have described similar, but more severe events in the eyes of patients with sickle cell hemoglobin-opathies.[2-5] The severity of the secondary glaucoma induced by bleeding of sickling erythrocytes into the anterior chamber has been emphasized by the occurrence of central retinal artery occlusion following mini-

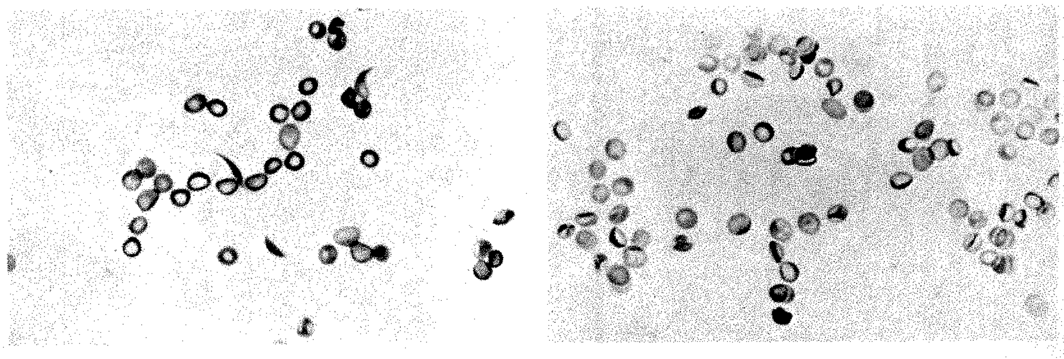

Fig. 4 (Goldberg). Case 2. Left, Erythrocytes from hyphema show more sickling than in venous blood from same patient. Right, Venous blood shows less sickling than in hyphema cells from same patient.

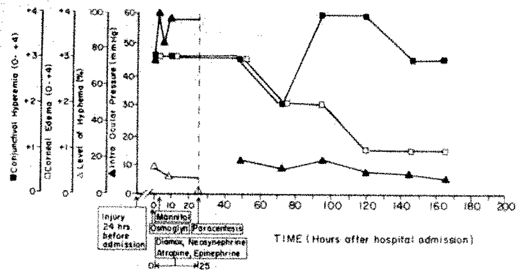

Fig. 5 (Goldberg). Case 3. Clinical course. Note inability of mannitol, glycerin (Osmoglyn), and acetazolamide (Diamox) to control intraocular pressure. Following paracentesis, pressure was normal (solid triangles).

mal hyphema formation in young individuals.[2-5] Because of the absence of other potential contributing factors, such as atherosclerosis, attention should be centered on the role of the sickled erythrocytes in causing sufficiently severe secondary glaucoma that the perfusion pressure in the central retinal artery is dangerously compromised.

Hyphema-induced secondary glaucoma and central retinal artery occlusion have occurred even in patients with sickle cell trait,[2-5] the most benign and the most common of the sickle cell hemoglobinopathies.

Because sickle cell trait is the most common of the sickle cell hemoglobinopathies, affecting at least 9% of the North American black population, the clinical management of hyphema in black patients deserves reappraisal. Clinical regimens for management of traumatic (or surgical) hyphemas are usually conservative in nature, because most hyphemas resolve without permanent complications.[6] The presence of sickled cells in the anterior chamber and trabecular meshwork, however, creates more serious conditions than in the normal patient incapable of having sickling in his erythrocytes. A high IOP of prolonged duration can reduce the intravascular perfusion in all intraocular tissues. With slowing of blood flow in the small vessels of the eye, including those of the optic nerve head, excessive deoxygenation of circulating erythrocytes will occur, thereby causing erythrostasis (stagnation leading to deoxygenation, to sickling, to rigidity of erythrocytes, to increased viscosity, to reduction of blood flow, to further stagnation).[7] An increase in IOP is more poorly tolerated by the eye of the patient with a sickle cell hemoglobinopathy than by the eye of a patient with normal red blood cells. A logjam of sickled erythrocytes is dangerous, not only within the trabecular meshwork, but also within the blood vessels of ocular tissues anatomically remote from the anterior segment.

Pharmacologic agents have been used

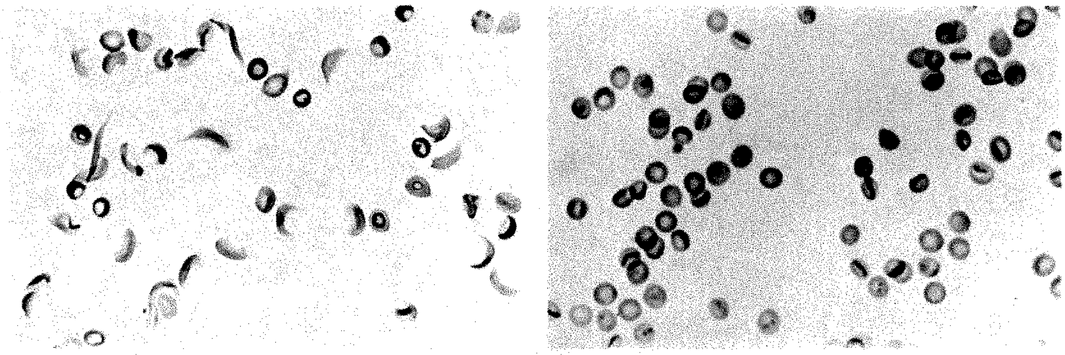

Fig. 6 (Goldberg). Case 3. Left Erythrocytes from hyphema show more sickling than in venous blood from same patient. Right, Venous blood shows less sickling than in hyphema cells from same patient.

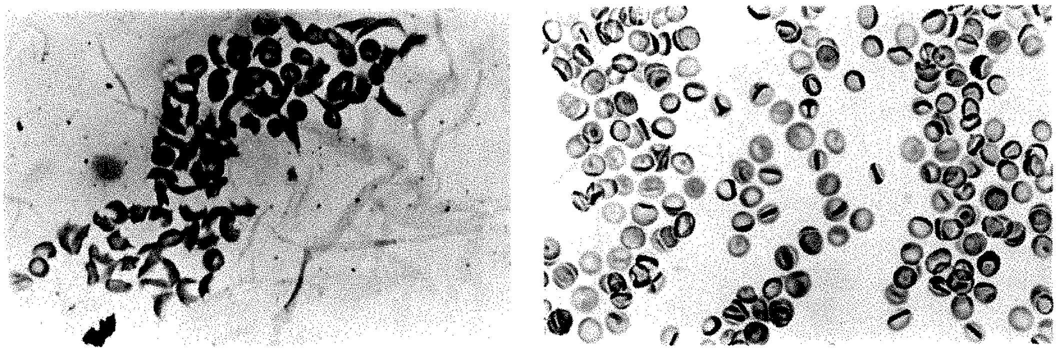

Fig. 7 (Goldberg). Case 4. Left, Erythrocytes from hyphema show more sickling than in venous blood from same patient. Right, Venous blood shows less sickling than in hyphema cells from same patient.

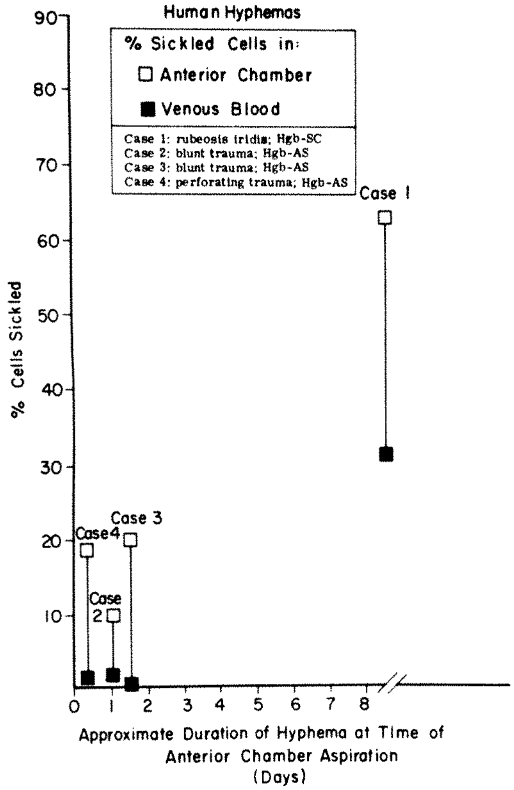

Fig. 8 (Goldberg). Percentage of erythrocytes found to be sickled in human hyphemas and in simultaneously obtained venous blood of the same patients. In all cases, the percentage of sickled cells was higher in aqueous humor than in the patients' venous blood.

for the management of hyphema-induced glaucoma in nonsickling patients. Most of these drugs have the potential for increased complications in patients capable of sickling. Repeated or excessive dosages of systemic dehydrating agents (that is, oral glycerin, intravenous mannitol, and diuretics) may increase hemoconcentration and viscosity in the already compromised ocular microvasculature (particularly in the central retinal artery,[3-5] the perifoveal vessels,[8] and the small vessels of the optic nerve head[8]). Carbonic anhydrase inhibition (with acetazolamide, for example) creates, in addition to hemoconcentration, the additional risk of systemic acidosis,[9] with its known tendency to exacerbate the sickling process.[10]

Methazolamide (Neptazane) may be a safer drug, because it contributes less to systemic acidosis than does acetazolamide, and because it may slightly increase the pH of the aqueous humor (whereas acetazolamide does not influence the normally acid pH of aqueous humor; B. Becker, M.D., written communication, July 20, 1977). That acetazolamide also increases the concentration of ascorbic acid in the aqueous humor[11] further discredits its clinical utility in this type of hyphema-induced glaucoma, because ascorbic acid can exacerbate the sickling process itself, presumably by acting as a reducing agent (unpublished observations).

These cases suggest that mannitol and acetazolamide can sometimes produce temporary reductions in IOP. Case 2 indicates (Fig. 3), however, that the favorable results may be short-lived (less than one to two hours), and as Case 3 indicates (Fig. 5), no therapeutic response may occur. Complications of therapy may ensue. A case has been reported of central retinal artery occlusion following mannitol administration to a young sickle cell trait patient who had glaucoma caused by blunt trauma and hyphema.[2] With these considerations in mind, one is left with virtually no therapeutic recourse wholly reliable for an eye with sickle cell hyphema and uncontrolled secondary glaucoma, except mechanical decompression and removal of some of the erythrocytes; that is, anterior chamber paracentesis or aspiration.

It has not been determined at what level IOP will irreversibly compromise vascular perfusion in the hemoglobinopathy patient. It has been suggested that flow in the central retinal artery of sickle cell patients may be seriously impaired at IOP levels higher than 40 mm Hg.[2] However, the small vessels about the macula and the small vessels in the optic nerve head[8] of the sickle cell patient may become relatively nonfunctional at even lower levels of IOP. Crouch and Frenkel[12] observed 27 patients with traumatic hyphemas. Only two patients in this group developed optic atrophy; both had sickle cell trait. In one patient the IOP varied between 35 and 39 mm Hg for only two days, and in the other patient it ranged between 35 and 40 mm Hg for four days. Paracentesis may be considered in this clinical situation when IOP is only slightly increased (perhaps 25 mm Hg or greater) for more than one day, despite use of antiglaucomatous drugs, and even if the hyphema is less than total. The relative benefits and risks of this seemingly aggressive surgical approach can be established by a prospective randomized assignment of patients to this regimen and, simultaneously, to a more traditional one.

Black patients with hyphema should have an immediate screening test for the presence of sickle hemoglobin, followed by a definitive hemoglobin electrophoresis. Intraocular pressure and visual function should be monitored several times a day. In the presence of a sickle cell hemoglobinopathy, heavy or repeated doses of acetazolamide and mannitol should be avoided, and early surgical removal of erythrocytes from the anterior chamber should be considered.

SUMMARY

Four patients with sickle cell hemoglobinopathies (one sickle cell hemoglobin C disease (SC); three sickle cell trait (AS)) and hyphemas had a higher percentage of erythrocytes sickled in their anterior chambers than in their circulating venous blood. Intraocular pressure (IOP) was severely increased, despite relatively small amounts of intracameral blood. Systemic hypotensive agents were not always successful in reducing IOP, and in patients with sickle cell hemoglobinopathy, are probably contraindicated in high or repeated dose regimens.

Moderate increase of IOP in sickle cell hemoglobinopathy patients may produced rapid deterioration of visual function, because of a greater than usual effect on vascular perfusion in the central retinal artery and optic nerve. Early anterior chamber paracentesis may be the best treatment for this type of hyphema-induced secondary glaucoma.

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OPHTHALMIC MINIATURE

"Then it's him I want to tell aboot it. T' Maister is blind o' the left eye."

"Nonsense!"

"It's true, sir. Not stone blind, but rarely fogged. He keeps it secret, but mother knows, and so do I. If thou slip him on the left side he can't cop thee. Thou'll find it right as I tell thee. And mark him when he sinks his right. 'Tis his best blow, his right upper-cut. T' Maister's finisher, they ca' it at t' works. It's a turble blow, when it do come home."

"Thank you, my boy. This is information worth having about his sight," said Wilson.

Sir Arthur Conan Doyle,
The Green Flag and Other Stories,
1900

TRANSCORNEAL FLUX OF TOPICAL PILOCARPINE TO THE HUMAN AQUEOUS

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Pilocarpine has been traditionally considered an alkaloid with differential solubility in water and lipids by reason of a polar-nonpolar molecular configuration, and it easily penetrates the cornea. The effectiveness of the drug in glaucoma control has been assumed to be based on this easy transport. Measurement of transcorneal flux of pilocarpine has rarely been discussed. The few reported assays have varied over a range of three orders of magnitude,[1-4] a disparity related to the methodological problems of measuring minute amounts of intact drug in a complex fluid such as aqueous.

Evidence suggests that conventionally applied pilocarpine penetrates poorly and the flux response to increasing doses does not show the linearity characteristic of simple diffusion transport. Increasing concentration of topical drops rarely improves response in proportion to increase in applied drug. This could imply site saturation, yet pilocarpine-soaked soft lens mediation is able to produce therapeutic responses much greater than those from topical drops.[5] This may suggest the presence of a corneal barrier, which is overcome by vehicular delivery to stimulate available sites. This interpretation seems hard to reconcile with the effect associated with the membrane-controlled delivery device of Alza (Ocusert), which mediates a therapeutic effect equalling that from pilocarpine drop doses higher by a whole order.[6] Because diffusion is concentration dependent, this effect is inconsistent with the assumption that pilocarpine diffuses easily across the cornea after drop administration or that hydrogel mediation simply mobilizes such a diffusion transport mechanism. Both hydrogel mediation and low dose membrane delivery enhance therapeutic response. The experimental rationalization of this paradox might help to clarify the mechanisms and limitations of conventional drop treatment with pilocarpine and similar alkaloids.

A series of in vitro experiments showed that high doses are poorly transported when pilocarpine is administered in conventional topical drops. Low doses are transported more efficiently by a mechanism mobilized by both hydrogel mediation and membrane controlled delivery. Isolated rabbit corneas in a transport chamber were used. To validate the concept as applicable to the human eye for glaucoma control, it was necessary to determine if pilocarpine in conventional high dose drop administration is poorly transported in vivo in the human eye, and if it is transported at rates consistent with the findings for the isolated rabbit cornea. We report herein experiments carried out to validate these points. Since the findings are significant particularly with reference to concepts developed from the in vitro experiments, these are presented first.

Most assays of flux have been made with use of taps from living eyes. The uncontrolled variables reduce the aqueous concentration of the active drug. These include eyelid action and tear dilu-

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This is an abridgment of a thesis submitted by Dr. Krohn as partial fulfillment of requirements for membership in the American Ophthalmological Society, May 1978.

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tion externally; and, within the eye, dilution by flow, protein binding, preferential binding to pigmented uveal tissue, degradation to pilocarpic acid or further, and epimerization to the inactive isopilocarpine. Aqueous pilocarpine concentration, as some measure of transcorneal flux, can be rendered more accurate by controlling the mechanical, chemical, and stereochemical variables. This was approached by development of a previously described[4] experimental chamber arrangement in which these internal ocular variables are largely excluded, but in which the main aspects of the physiological situation, except for eyelid action, are maintained in a standardized manner.

The effect on transcorneal flux of pilocarpine was measured in this chamber system with various modes of delivery. The findings[4,7,8] can best be expressed in terms of flux efficiency (transport × time^{-1}/dose).

A typical drop dose showed a low flux efficiency (Fig. 1) of about 0.12%. Accumulation of this fraction in the aqueous chamber analog continued over a 90-minute period, but measurements of tear analog efflux indicated that all transported drug had been taken up by the cornea within ten minutes of administration (Fig. 2). The drug was determined by ultraviolet spectrophotometry. In this closed system, 30% of the drop-administered dose was lost within the cornea. This fraction could not be eluted as intact pilocarpine from the cornea by further flow of tear analog. For this reason, we have called it a corneal depot as opposed to a reservoir (Fig. 2).

In contrast, as seen in Figure 1, drug transport in hydrogel polymer vehicles continued until the vehicle was completely eluted of its pilocarpine, a time inversely related to the resistance of the particular vehicle to tear elution (Fig. 1). Despite this variation to resistance to elution by these respective polymer preparations with the consequent large difference in the rates at which drug was eluted into the tear flow, the transcorneal flux slopes of all four vehicles were congruent to the point at which each vehicle, in turn, was completely eluted. Within the limits of the rate of elution allowed by the physical factors of all four vehicles, flux was thus independent of dose. This finding was further supported by available dose measurements at various intervals for a 55% hydrated lens vehicle (Fig. 2, area above upper curve). This decreased exponentially, whereas transcorneal flux

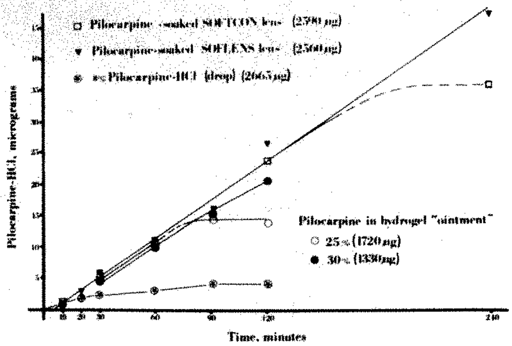

Fig. 1 (Krohn and Breitfeller). Transcorneal flux of pilocarpine HCl in vitro after delivery in polymer gel (25% and 30%), hydrogel lens buttons, and in free fluid (simulated drops). Mean doses in parentheses.

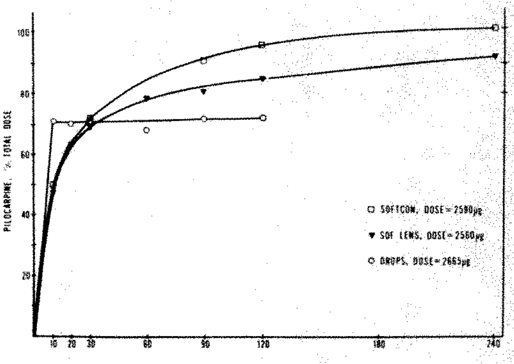

Fig. 2 (Krohn and Breitfeller). Pilocarpine HCl in effluent tear analog after delivery by pilocarpine soaked hydrogel polymer lens buttons and pilocarpine solution.

mediated by the same vehicle was linear to the point of elution (Fig. 1). The depot phenomenon seen with the single dose fluid drop administration was absent because the total drug recovered equalled the administered dose (Fig. 2). Finally, both total transport and flux efficiency increased up to ten times relative to drop administration.

In continuous flow pilocarpine administration via the tear analog without vehicle interposition, a single time interval of 90 minutes was used and the dose varied. Doses in standard pharmacological range were transported as a function of dose. Flux efficiency was only modestly improved over that found for equivalent dosage in drops. As dose decreased, flux efficiency increased as an inverse function of the log of the dose (unpublished data). This series of experiments seemed to indicate that high doses are inefficiently transported, whether administered as the typical milligram-level bolus in common clinical use or in constant flow.

These findings were for uniform corneal preparations from enucleated albino rabbit eyes. To support these data, it was necessary to test some measurable factor that could establish a correspondence between the function of a rabbit cornea in an artificial chamber and the human eye.

Taps of human aqueous at the time of cataract extraction after typical pilocarpine drop doses presented such an opportunity, despite the uncontrolled variables. An extremely low concentration limit would have to be found if chamber investigations and concepts derived from them are consistent with human transcorneal flux. Determination of pilocarpine in the minute quantities anticipated to be found in these taps required the security of two separate analytical methods.

MATERIAL AND METHODS

Aqueous pilocarpine assay by ferric hydroxylamine complexing—The aqueous fluid that is normally lost to the field in ordinary cataract extraction was collected with informed consent and replaced by saline before corneoscleral section. The eyes were randomly selected as to age, race, or underlying disease in two groups, the first consisting of controls. In a second group, two drops of 2% pilocarpine HCl in a standard buffered and preserved commercial preparation were placed onto the apex of the cornea under general anesthesia. After routine preparation for surgery, but exclusion from the sac of any further solution, copious irrigation was followed immediately by careful drying and aqueous collection. The influence of drug or serum contamination or blinking were excluded. Elapsed time between pilocarpine application and both irrigation and tapping were recorded. Pilocarpine was determined in the collected samples by optical absorption of a ferric hydroxylamine complex at 540 nm in a Zeiss spectrophotometer by the method of Hestrin.[9] A reagent blank and pilocarpine standards, using saline as diluent, were included for each run.

The method was tested for linearity in saline and pooled rabbit aqueous. The extreme deviation from the predicted regression line in any of the 20 standard curves represented less than 2 μg of pilocarpine. Although the curves for pilocarpine in pooled primary rabbit aqueous and saline are parallel (Fig. 3), they are not congruent, because the color complexing is not specific for pilocarpine but will develop with various other constituents of aqueous fluid accounting for up to 70% of the color complex. Concentrations of these reacting substances will vary considerably from eye to eye and from time to time. A determination of drug appearing in the chamber after drop instillation therefore required a sufficient number of samples in each category for any increment in optical absorption due to pilocarpine to be statistically significant.

Aqueous pilocarpine assay by gas-

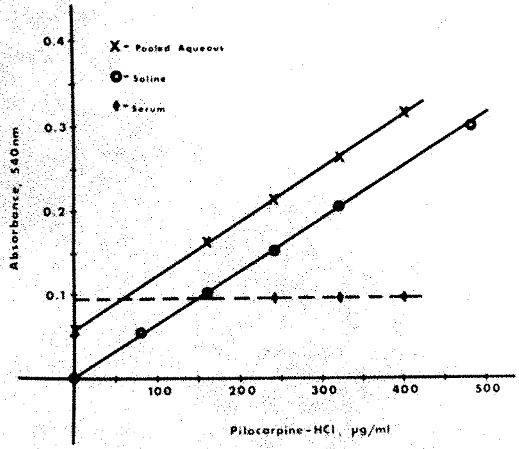

Fig. 3 (Krohn and Breitfeller). Standard curves for pilocarpine HCl in pooled rabbit aqueous fluid, normal saline, and rabbit serum by optical density of a ferric hydroxylamine-pilocarpine complex at 540 nm.

liquid chromatography—The limitations of available techniques, including that previously described, prompted a search for a better method to determine pilocarpine in aqueous fluid. A gas-liquid chromatographic method was developed[10] which is highly specific and accurate to subnanogram levels. The technique avoids the cross protonation and radiolysis problems inherent in methods using tritium-labelled drug. The small volume of aqueous required by this method enabled the technique of collecting samples to be completely noninvasive.

Participants and doses were as previously described. After copious irrigation and careful drying, the initial corneoscleral section of approximately 4 mm was made without loss of aqueous fluid. The tip of a 100-μl micropipette, appropriately bent for convenience of application, was approximated to the incision without pressure, and between 25 and 35 μl of aqueous withdrawn slowly by capillarity only. Intervals between instillation of drops and both pretap irrigation and aqueous withdrawal were recorded.

Pilocarpine concentration in the aqueous samples was determined by gas-liquid chromatography with electron capture detection of a heptafluorobutyryl derivative. Recovery of added pilocarpine was 95% from both saline and pooled rabbit aqueous. A total of 71 experimental and 11 control samples were analyzed. Additionally, a control series was done for similar taps in albino New Zealand rabbits, because the time intervals could be more deliberately distributed.

RESULTS

Aqueous pilocarpine assay by ferric hydroxylamine complexing—The means for aqueous concentration of reacting substances were almost identical in the control and experimental samples (Table). There was no difference between early and late taps. The limit of similarity between control and experimental groups, using a putative P value of .05 would be reached if the difference between the two mean averages were 22 μg/ml. Since the method of determination itself is highly reproducible, this statistical limitation is the result of the variation in background complexing substances in the individual aqueous samples. The data indicate that under the circumstances described, chamber concentration after a dose of about 2,000 μg of pilocarpine HCl

TABLE

AQUEOUS PILOCARPINE BY HESTRIN REACTION

	No. Determined	Mean, μg/ml	SD	SEM	*t*-Test
Control	21	205	53	12	
Treated	23	204	33	7	P>.05 (0.8)

in drops was 22 μg/ml or less within 25 minutes after instillation. This worst case represents a flux efficiency of about 0.5%. However, the close agreement of the means suggests that the actual concentration of pilocarpine and the flux efficiency were both much lower.

Aqueous pilocarpine assay by gas-liquid chromatography—Control human aqueous showed no evidence of pilocarpine by this method. Experimental determinations, plotted in Figure 4, show a clustering of points in the interval between six and 26 minutes after instillation. This occurred because these intervals had to be governed by surgical considerations only. At no time following the topical instillation of approximately 2,000 μg of pilocarpine was aqueous concentration higher than 5 μg/ml. Since aqueous volume is under 400 μl, the highest level for total aqueous pilocarpine was in the range of 2 μg. The mean concentration for the series was 1.67 μg/ml, with a mean total aqueous pilocarpine estimated to be approximately 0.67 μg/ml representing a flux efficiency of about 0.03%. The highest concentration for this series was found at about ten minutes after instillation. The apparent limit of net aqueous pilocarpine was 0.5 $\mu g \cdot ml^{-1} \cdot min^{-1}$. Distribution of findings for taps in eyes of rabbits (Fig. 5) was similar, although the highest concentration found was 9 μg/ml, and the interval at which highest concentration appeared was some six minutes later than in the eyes of cataract patients.

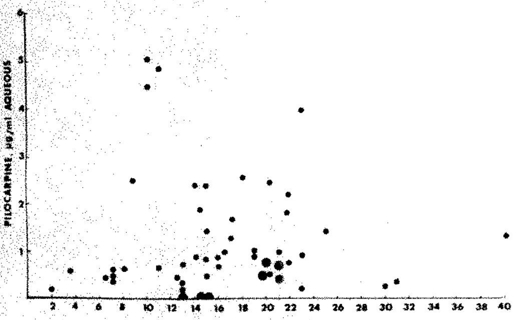

Fig. 4 (Krohn and Breitfeller). Aqueous concentration of pilocarpine HCl in human anterior chambers at various intervals following topical instillation of two drops of 2% solution. Nominal dose, 2,000 μg.

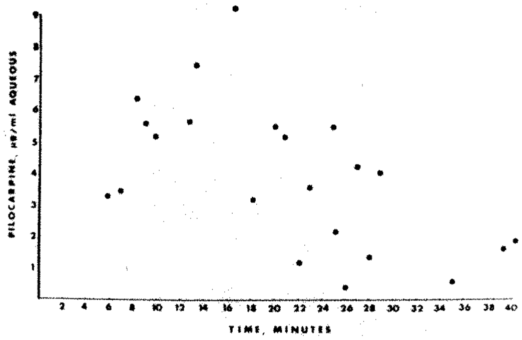

Fig. 5 (Krohn and Breitfeller). Aqueous concentration of pilocarpine HCl in anterior chambers of 2 to 3-kg albino New Zealand rabbits at various intervals following topical instillation of two drops of 2% solution. Nominal dose, 2,000 μg.

DISCUSSION

The gas-liquid chromatography findings correspond with those found to be statistically valid in the series determined by colorimetry. The latter method was far less sensitive but indicated a significant difference from the lowest level of previous flux estimates and supplied a second method control for the gas-liquid chromatography measurements. The remarkably low flux efficiency found with both analytical techniques supports the validity of the chamber experiments as applicable to the living human eye. Apparent flux efficiencies in tap experiments were less than that found in the chamber experiments, averaging approximately 0.03%. However, in the chambers, external and intraocular events, which all tend to reduce drug concentration, were deliberately excluded. The human tap findings, expressed in terms of flux efficiency, were less than half those of Chrai and Robinson[11] for rabbits, and 20 times lower than those of Asseff and associates[2] for primates. Both studies used tritiated pilocarpine. Although the time course of drug appearance in the aqueous differed markedly from that found by Asseff and associates,[2] it was similar to that reported by Harris.[3]

The combined laboratory and human tap data seem to indicate that pilocarpine behaves as a poorly lipid soluble molecule that, in high doses (>3 ymol/hr), is translocated from the tear film to the aqueous chamber slowly and inefficiently by a dose-dependent mechanism. The substantial portion rapidly absorbed into the cornea is complexed or degraded to products, still undetermined, which probably lack cholinergic effect. A small fraction of the intracorneal moiety eventually diffuses to the aqueous. In contrast, vehicle mediated transport has up to ten times higher flux efficiency and is capable of producing substantially higher absolute yield of intact drug to the aqueous than that available from drop regimens, both experimental and clinical. Doses available by vehicle mediation appear to be transported at a rate determined by the cornea and independent of dose, kinetics suggesting the possible utilization of some type of saturable carrier mechanism. Since the nonelutable depot is associated with high dose and inefficient transport, it is consistent that the conformation of such a putative carrier is altered by high concentration of pilocarpine that allows complexing to secondary binding sites of low affinity. Such nonspecific tight binding of pilocarpine to protein might explain the apparent poisoning of the saturable low dose transport mode. The depot may represent in large part complexed or degraded pilocarpine, or both, which is diffusing, or has already diffused away.

The function of a transport-enhancing vehicle is to retain drug not only against mechanical dissipation and tear flow elution, but against the capability of the cornea to take up more drug than it can transport intact. The excess seems to disable the low dose mechanism. Enhanced transport depends in part on prolonged mobilization of the efficient low dose mechanism to its useful limit. The constant flow experiments suggest that low dose and high dose mechanisms are operative at any dose, but the net effect is a log function of dose. Transport enhancement by hydrogels was greater than can be attributed only to continuously applied low dose, possibly because of opportunistic recruitment of carrier mediation as suggested by the kinetics of drug delivery by soft lens vehicles.

SUMMARY

Aqueous fluid was withdrawn from eyes of patients undergoing cataract extraction at various intervals after administration of two drops of 2% pilocarpine HCl in a standard manner. Determination of aqueous pilocarpine concentration was made both by spectroscopy of a ferric hydroxylamine complex and by gas-liquid chromatography. Results of both methods were consistent in indicating that concentration does not rise at any time following such topical instillation beyond 5 μg/ml, with an average of 1.67 μg/ml, representing a flux efficiency of 0.03%.

These findings correlate well with previous investigations of transcorneal flux of pilocarpine for the rabbit in a transport chamber system, in which comparable low flux efficiency was found after simulated drop administration. This serves in some measure to validate an extrapolation of other findings in chamber experiments to the living human eye.

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OPHTHALMIC MINIATURE

Instances of acuteness of sight are to be found stated, which, indeed, exceed all belief. Cicero informs us, that the Iliad of Homer was written on a piece of parchment so small as to be enclosed in a nutshell. He likewise mentions a man who could distinguish objects at a distance of one hundred and thirty-five miles. M. Varro says, that the name of this man was Strabo; and that, during the Punic war, from Lilybæum, the promontory of Sicily, he was in the habit of seeing the fleet come out of the harbor of Carthage, and could even count the number of vessels. Callicrates used to carve in ivory ants and other small animals, so minute that other persons were unable to distinguish their individual parts. Myrmecides also was famous in the same line; this man produced, of similar material, a chariot drawn by four horses, which a fly could cover with its wings; as well as a ship which might be covered by the wings of a tiny bee.

Casey A. Wood, editor, *The American Encyclopedia and Dictionary of Ophthalmology*
Chicago, Cleveland Press, 1918

DELAYED ABSORPTION OF SUBRETINAL FLUID AFTER SCLERAL BUCKLING PROCEDURES

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The recent more common usage of Custodis-type nondrainage procedures in the management of rhegmatogenous retinal detachments has required additional decisional expertise in judging which cases might be amenable to nondrainage techniques and which might be managed more appropriately with release of subretinal fluid. After either type of procedure, proper assessment of the absorption of subretinal fluid during the postoperative period is important.

Whereas abnormal persistence of subretinal fluid may indicate the presence of small unidentified and untreated holes and the need for additional surgical treatment, in certain circumstances subretinal fluid may persist for an unusually long time before being totally absorbed. In such circumstances, the retinal breaks may have been treated adequately, but the mechanism for resorbing subretinal fluid may have been deficient. It is important that the ophthalmologist be familiar with the clinical characteristics of such cases to avoid unnecessary reoperation.

I describe herein the preoperative clinical characteristics of rhegmatogenous retinal detachments that are associated with delayed absorption of subretinal fluid in a group of patients treated by means of scleral buckling. In selected patients, subretinal fluid collected at operation was examined for protein or cellular characteristics in an effort to provide information that might aid in the explanation of delayed absorption of fluid. For this report, delayed absorption of subretinal fluid is defined as the condition existing when subretinal fluid persists longer than six weeks after a surgical procedure for rhegmatogenous retinal detachment, but ultimately absorbs completely with total retinal detachment.

MATERIAL AND METHODS

Of 598 consecutive cases from 1970 through 1976 in which I used scleral buckling to treat rhegmatogenous retinal detachment, six-month follow-up data were available for 575 cases, 92% of which were ultimately successfully reattached. Information was prospectively collected and recorded on special, coded questionnaire forms used throughout the study. Cases were analyzed with respect to the interval between operation and total absorption of fluid. Those cases in which absorption was delayed longer than three months and those in which absorption was delayed between six weeks and three months were studied separately to learn if common preoperative, operative, or postoperative factors that might be associated with prolonged fluid absorption could be identified.

CASE REPORT

A 67-year-old woman came here with a two-week history of visual blurring in the left eye. Visual acuity was 6/6 (20/20) in the right eye and finger counting in the left eye. A one-quadrant detachment containing a single horseshoe tear was present in the superior temporal quadrant of the left eye. The detachment was not unusual, except for the presence of numerous small cream-colored lesions, similar in appearance to mutton-fat keratic precipitates, located on the undersurface of the detached retina (Fig. 1). Because they were on the back surface of the detached retina, I referred to them as subretinal precipitates.

The detachment was treated with a segmental

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This is an abridgment of a thesis submitted by Dr. Robertson as partial fulfillment of requirements for associate membership in the American Ophthalmological Society, May 29, 1978.

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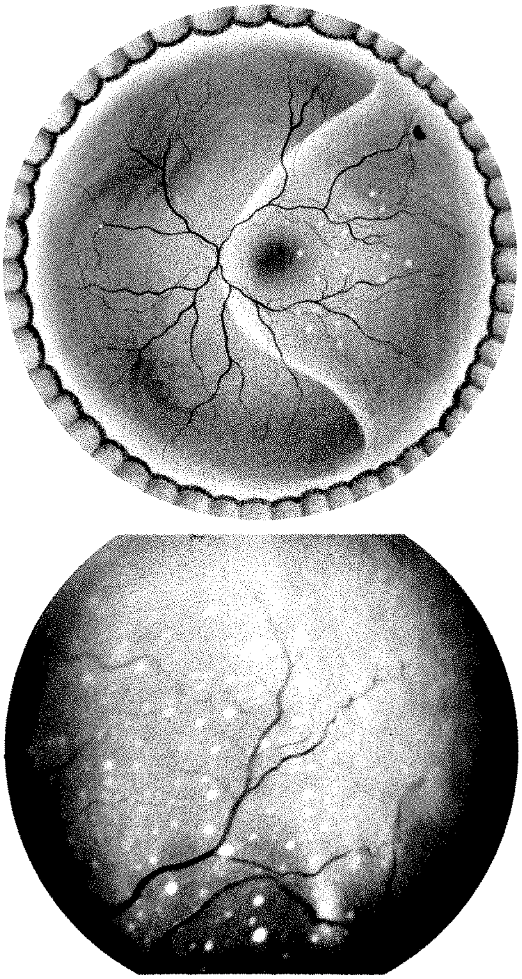

Fig. 1 (Robertson). Top, Schema of retinal detachment showing single small horseshoe tear and relative distribution of subretinal precipitates. Bottom, Retina of same patient showing numerous dotlike subretinal precipitates.

buckling procedure using cryopexy and an episcleral sponge without release of subretinal fluid. Within three days, the subretinal fluid seemed to have absorbed from the posterior pole. However, one month later the distribution of the subretinal fluid had changed little, if at all. To eliminate the possibility that fluid was leaking through the treated tear or through another unrecognized, untreated break, the buckle was revised with a second nondrainage surgical procedure to make it broader and higher.

Four months after the second surgical procedure, subretinal fluid was still present. However, when the patient returned as instructed, 11 months after the second operation, the retina was totally attached and the visual acuity had recovered to 6/9 (20/30).

RESULTS

Since subretinal fluid persisted for months after the second surgical procedure, I concluded that revision of the buckle probably had been unnecessary. This case made me curious about the significance of subretinal precipitates. After seeing additional rhegmatogenous detachments with subretinal precipitates, I soon formed the clinical impression that if subretinal precipitates could be recognized easily before operation by indirect ophthalmoscopy, a lengthy delay in absorption of subretinal fluid could be anticipated in some cases after scleral buckling, irrespective of partial drainage of the subretinal fluid.

A second condition that seemed important to analyze in this study was the presence or absence of subretinal precipitates before operation (Fig. 2). The cases with subretinal precipitates were studied to determine the relationship between subretinal precipitates and a delay in absorption of subretinal fluid after scleral buckling.

In 19 patients, subretinal fluid was not absorbed ultimately for three months or more (Table 1). In nine of the 19 patients, the preoperative examination identified the presence of subretinal precipitates. In seven of the remaining ten patients, the detachments were caused by small, round peripheral atrophic holes without opercula or evidence of vitreous reaction. Of the other three patients, one had a longstanding inferior dialysis, one had complications from five previous operations, including an unsuccessful attempt to repair the retinal detachment, and one had had previous trauma to the eye with intraocular hemorrhage and an unsuccessful attempt to repair the retina one month earlier at another institution.

In 15 of these 19 patients, subretinal fluid was released through a sclerotomy, although no special effort was made to drain all of the subretinal fluid.

Most of the nine patients with preoper-

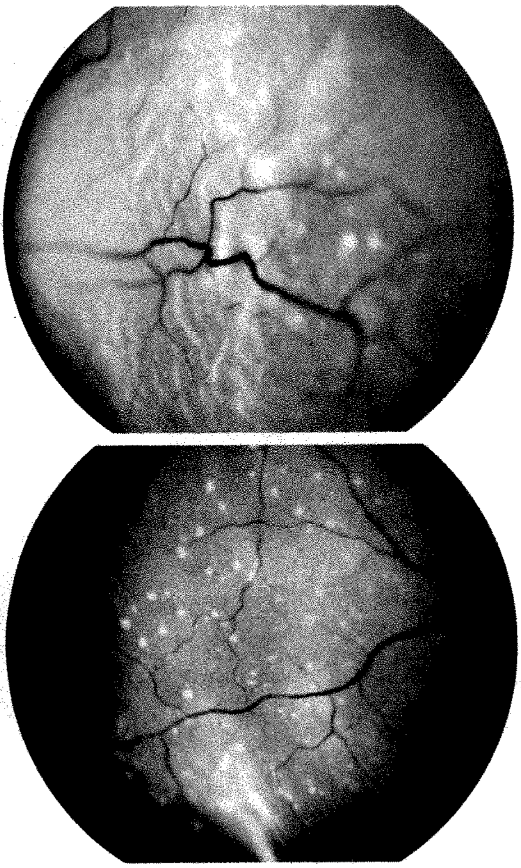

Fig. 2 (Robertson). Clinical appearance of retinal detachments with subretinal precipitates. Top, Moderate numbers of precipitates. Bottom, Large numbers of precipitates.

ative subretinal precipitates had fresh detachments, that is, detachments of recent onset. In six of the nine patients, the duration of the detachment was estimated to be between five days and three weeks. Two of the nine detachments were caused by small round peripheral holes, whereas the others were caused by horseshoe tears. In seven of the nine patients, subretinal fluid was drained at the time of operation.

TABLE 1
ABSORPTION OF SUBRETINAL FLUID DELAYED BEYOND THREE MONTHS (19 CASES)

Characteristics of Retinal Detachments	No. of cases
Subretinal precipitates	9
Small round atrophic holes	7
Inferior dialysis	1
Complicated case	2

In contrast to the detachments associated with subretinal precipitates, those in the seven patients having small round atrophic holes without subretinal precipitates were generally of long standing. In five of the seven patients, for example, demarcation lines were observed. In two of the seven patients the detachment involved the macula. In six of the seven detachments, subretinal fluid was drained. Three of the seven patients had lattice degeneration (Fig. 3).

A collective analysis of all the cases in which subretinal fluid persisted longer than six weeks, including those with fluid persisting longer than three months, revealed a broader variety of retinal detachment (Table 2). Of the 39 patients in this entire group, 17 (44%) had subretinal precipitates; in 14, subretinal fluid was drained. In 11 additional patients without subretinal precipitates, detachment was associated with peripheral atrophic round

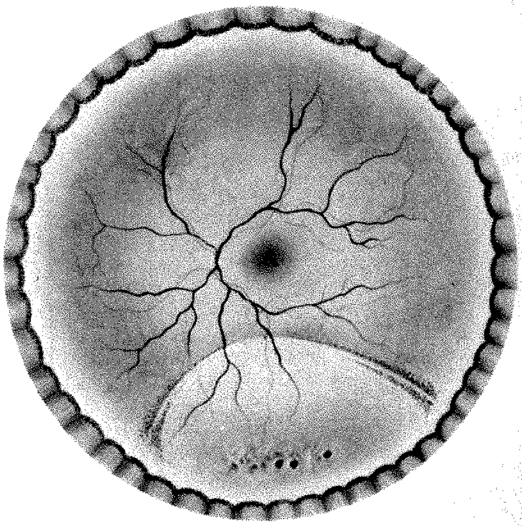

Fig. 3 (Robertson). Schema of retinal detachment caused by round peripheral atrophic hole. Note pigmented demarcation lines.

TABLE 2

ABSORPTION OF SUBRETINAL FLUID DELAYED MORE THAN SIX WEEKS (39 PATIENTS)

Characteristics of Retinal Detachment	No. of cases
Subretinal precipitates	17
Small round atrophic holes	11
Exudative detachment	3
Other long-standing detachments	2
Subretinal hemorrhage	1
Complicated case	3
Vitreous changes	2

holes (six had demarcation lines, and six had lattice degeneration). Among all 39 cases, delayed absorption of fluid was unrelated to the age of the patient, size of the largest break, number of cryopexy lesions, number of quadrants of detached retina, presence or absence of aphakia, and presence or absence of uveitis.

The distribution of the final visual acuities in the 39 patients with delayed fluid absorption was not significantly different from the distribution in the overall series.

Of the 575 cases, 69 (12%) had subretinal precipitates (Table 3). Of these 69 cases, 17 (25%) were associated with a delay in complete absorption of subretinal fluid for an interval longer than six weeks.

Laboratory studies were conducted on subretinal fluid collected from 43 patients. Analysis was done only in those patients in whom the sclerotomy for drainage of subretinal fluid was conveniently located for collection of fluid. After sclerotomy, 0.25 to 0.5 ml of subretinal fluid was collected; if the fluid was visibly contaminated by blood, the samples were discarded and further efforts to collect fluid were abandoned. The subretinal fluid was analyzed for total protein in 39 cases.

TABLE 3

RELATIONSHIP OF SUBRETINAL PRECIPITATES TO CASES WITH DELAYED ABSORPTION OF SUBRETINAL FLUID

Incidence	
In 575 cases	69/575 (12%)
In cases with fluid absorption delayed six weeks	17/39 (44%)
Statistical significance of relationship of precipitates to delayed fluid absorption as determined by chi-square test	P<.001

In 18 of the 39 patients, subretinal fluid was collected before cryopexy to avoid disruption of the chorioretinal complex (exudation and so forth) by cryopexy and the consequent alteration of the subretinal fluid composition. In three patients, samples were collected before and after cryopexy.

In three cases the cellular content of samples of Millipore-filtered subretinal fluid was stained by a modified Papanicolaou technique. In one additional case in which subretinal precipitates were plentiful, the subretinal fluid was studied by electron microscopy. In each of these four cases studied for cellular content, the fluid was collected before cryopexy.

A positive relationship could not be recognized between the protein concentrations and any of the following factors: the patient's age; the duration of the detachment; the number of quadrants involved; the presence or absence of aphakia, demarcation lines, and subretinal precipitates; the interval between surgery and resorption of fluid; the size of the largest break. However, a positive relationship existed between the protein concentration and the estimated duration of the detachment ($P = .05$ by the chi-square test). In three instances, a protein analysis of subretinal fluid was made, both before and after cryopexy, to determine if significant alterations in protein concentrations were induced. No significant differences were recognized despite extensive cryopexy (for example, in one case, 70 freezes were used). The highest protein concentration was found in a patient in whom the estimated duration of the detachment was longer than one year. The second highest concentration was found in a patient undergoing reoperation.

Structural elements obtained before cryopexy consisted of red blood cells, pigment debris, pigment-laden macrophages, and occasional macrophages without significant pigment. Pigment-laden macrophages were particularly plentiful in one of the cases with large numbers of subretinal precipitates (Fig. 4). Electron microscopic analysis of ultracentrifuged subretinal fluid in the one case studied showed red blood cells, extracellular cytoplasmic debris, and macrophages, some with ingested melanin (Fig. 5, top) and some in which abundant pigment granules were found free in the cytoplasm (Fig. 5, bottom).

DISCUSSION

Hofmann and Hanselmayer[1] learned that spontaneous flattening of retinal detachments, by means of preoperative immobilization of the patient, was most likely to occur in fresh detachments, in young persons, and in detachments in which the breaks were relatively large (approximately one disk in diameter). Inferior detachments, aphakic detachments, and those with either small or large breaks were less likely to flatten. In a review of 200 cases, Leaver and associates[2] noted persistence of subretinal fluid beyond one week in 25% of the patients treated by nondrainage techniques. The only definite factor they could recognize relating to the rate of absorption of subretinal fluid was the duration of the retinal detachment; no relationship was found between the rate of absorption and

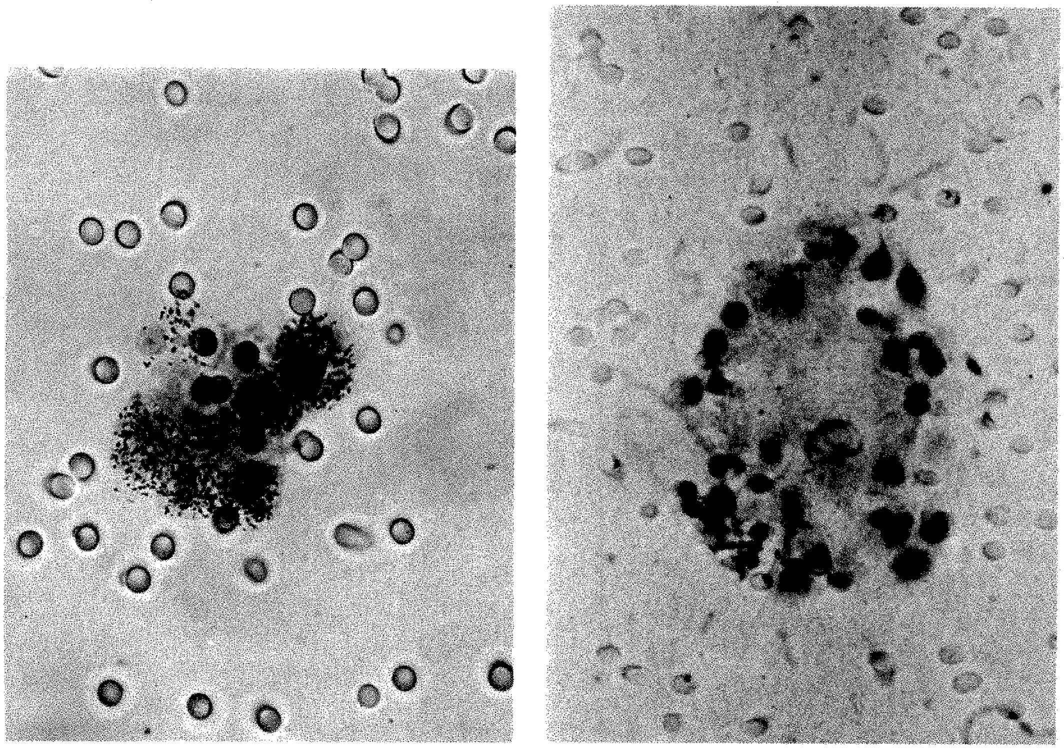

Fig. 4 (Robertson). Left, Pigment-containing macrophage from filtered subretinal fluid stained by Papanicolaou technique. The discrete circles represent pores in Millipore filter. These pores are 5 μm in diameter. Right, Free macrophage containing minimal pigment (×400).

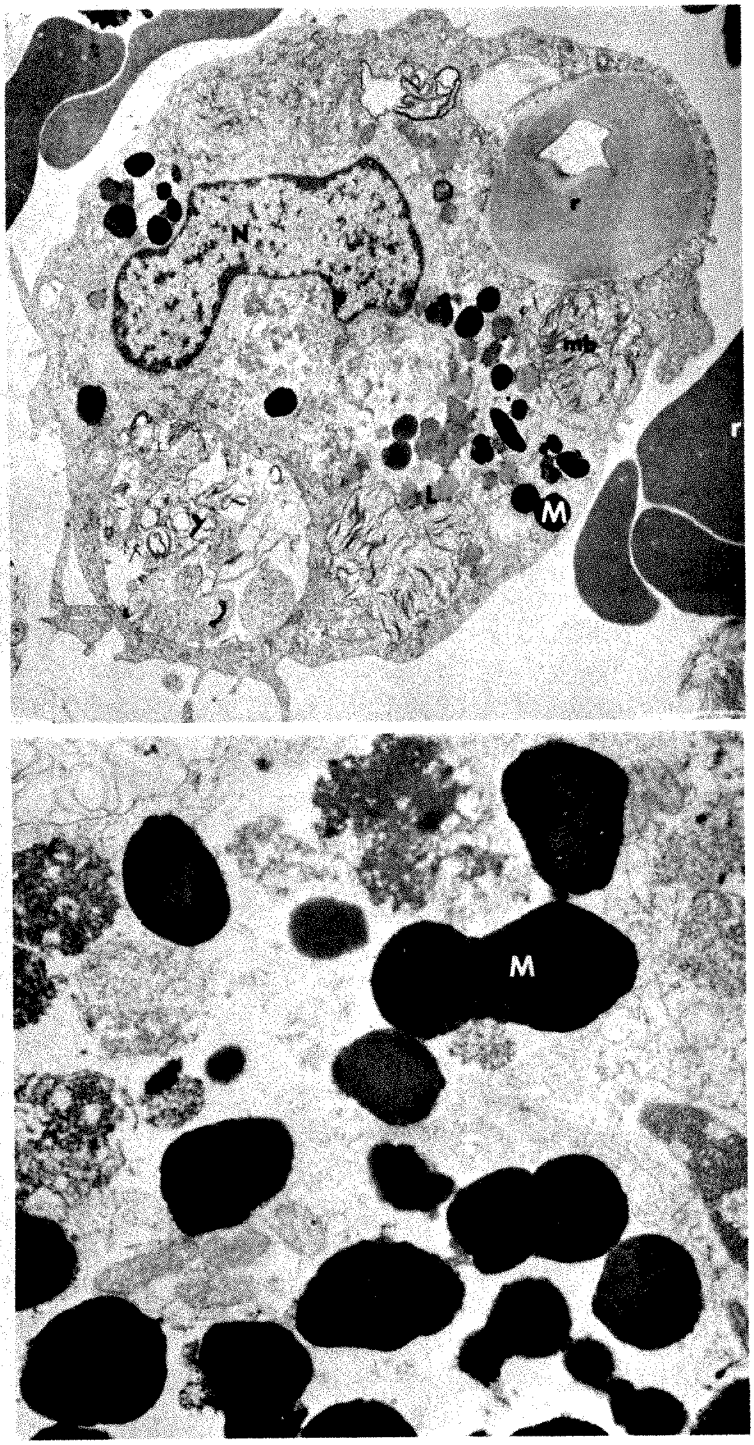

Fig. 5 (Robertson). Top, Macrophage with ingested melanin granules (M), lipid particles (L), membranous bodies (mb), and red blood cell (r); nucleus (N). (×4,500). Bottom, Cell with abundant melanin granules (M) (×10,000).

age of the patient, severe myopia, or volume of subretinal fluid. Their findings were at variance with those of Nadel, Gieser, and Lincoff[3] and of O'Connor,[4] who concluded that age correlated positively with delayed absorption of subretinal fluid.

I analyzed only those cases in which subretinal fluid persisted beyond six weeks before ultimately absorbing completely with total reattachment. Within this time interval, analysis of all the data failed to show a positive relationship between delayed fluid absorption and the age of the patient, number of quadrants detached, size of the breaks, the presence or absence of aphakia, or the number of cryopexy lesions used, although a positive relationship was identified relative to the estimated duration of the detachment. A heretofore unreported relationship was recognized between delayed absorption of fluid and the presence of subretinal precipitates that were observed before operation.

In this study the single most common preoperative clinical characteristic associated with a delay in subretinal fluid absorption was the presence of subretinal precipitates. The relationship between the presence of subretinal precipitates and the delayed absorption of fluid is highly significant statistically ($P < .001$) (Table 3). If these precipitates are recognized before operation, statistical analysis indicates that the clinician can expect approximately one fourth of the patients to exhibit a delay in complete absorption of subretinal fluid of more than six weeks. Knowing that subretinal precipitates were present before operation should discourage operations unless clear evidence of other singular causes of retinal detachment exist. This study did not provide evidence to indicate that patients with retinal precipitates should necessarily undergo drainage; those who did not undergo drainage seemed to do equally well in terms of final visual acuity and reattachment. In the follow-up of patients with subretinal precipitates, I observed that the peripheral retina appeared to reattach slowly and usually only after subretinal precipitates had disappeared.

I do not know the precise role of the subretinal precipitates, but their occasional presence in rhegmatogenous retinal detachment has been recognized clinically since 1938 when a single case was reported by Vogt.[5] On the basis of their resemblance to mutton-fat keratic precipitates, the histologic studies in human eyes reported by Zimmerman,[6] and what is known of the histology of experimental detachments in the owl monkey,[7] these precipitates are presumably accumulations of macrophages. This observation is supported further by the light microscopic finding of macrophages and pigment epithelial cells in human subretinal fluid studied by Lam and associates,[8] and by the finding of many macrophages in the subretinal fluid of patients in this study identified as having subretinal precipitates before operation. Electron-microscope examination of the cells collected in advance of treatment in one case indicated that in some of the cells melanin pigment granules were freely present in the cytoplasm rather than within phagosomes; this finding suggests that some cells actually may be retinal pigment epithelial cells. These electron-microscope findings are consistent with the electron-microscope findings in experimental retinal detachment in the owl monkey reported by Machemer and Laqua.[9] They concluded that the pigmented single or clustered cells that acted as macrophages derive from the pigment epithelium. Electron-microscope studies of cellular components of human subretinal fluid in rhegmatogenous retinal detachments also have been reported by Feeney and associates.[10]

Although the cells that were recovered from subretinal fluid samples in this study were not obtained from direct aspiration of subretinal precipitates, the supposition that the subretinal precipitates are composed of similar cells is strong, based on studies in the animal eye. The role of these macrophages may be to phagocytose degenerating outer segments of the photoreceptors or the mucopolysaccharides, which are normally present between each of the outer cell segments and the space between the photoreceptors and the retinal pigment epithelium. I cannot explain why they seem to be abundant in some cases and infrequent in others. In the few cases with subretinal precipitates that I have subjected to subretinal fluid protein analysis, I have found that the protein concentrations were variable, but in no instance were they unusually high.

The second most common condition in this study associated with a delay in subretinal fluid absorption was a long-standing peripheral retinal detachment (usually located inferiorly), which generally spared the macula, was associated with demarcation lines, and was caused by round atrophic holes with or without associated lattice degeneration. When the cases with demarcation lines were analyzed, the relationship between the presence of such lines and the presence of delayed fluid absorption was statistically higher than their frequency in the overall series ($P < .02$). This finding indicates a positive relationship between long-standing retinal detachments (identified by presence of demarcation lines) and delayed absorption of subretinal fluid.

SUMMARY

Delayed absorption of subretinal fluid beyond six weeks after surgical repair for rhegmatogenous retinal detachment was recognized in 39 of 575 consecutive cases of scleral buckling. The most common preoperative condition was large clumps of cells on the undersurface of the detached retina (subretinal precipitates). Approximately one in four patients in whom precipitates are seen preoperatively will have fluid persisting beyond six weeks from surgical repair to complete absorption.

A second relatively common condition associated with delayed fluid absorption that could be recognized before operation was long-standing peripheral (usually inferior) retinal detachment, which typically spared the macula, was associated with demarcation lines, and was caused by round atrophic holes with or without associated lattice degeneration.

An analysis of subretinal fluid protein concentrations in 39 cases showed a positive relationship between protein concentration and duration of detachment. Pigment-laden macrophages in the subretinal space, possibly originating from the retinal pigment epithelium, were common.

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THE TREATMENT OF ACUTE BRANCH VEIN OCCLUSION BY PHOTOCOAGULATION

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Retinal branch vein occlusion was first observed and described by Leber in 1888. Although widely studied, it continues to challenge therapeutic attempts at preservation and restoration of vision.[1]

Branch vein occlusion may be divided into two phases, acute and chronic. The acute phase is characterized by a sudden onset of visual disturbances often involving the macula. Various degrees of venous obstruction occur and this obstructive vascular stage is accompanied by retinal hemorrhages, exudates, and edema in the area distal to the obstruction. A few flame-shaped hemorrhages may occur with minimal retinal edema and exudates. With more extensive obstruction, there may be marked retinal elevation with extensive retinal hemorrhage obscuring all the retinal structures. The macula may be totally spared, moderately involved with edema, or totally obscured by retinal edema and hemorrhages.

Fluorescein angiography reveals obstruction of the venous circulation at the site of the arteriovenous crossing as seen by delayed emptying of the retinal veins distal to the occlusion.

Paravenous leakage of fluorescein dye and diffuse late leakage of dye into the adjacent tissue is visible (Figure, top left). The angiographic picture may be totally obscured by the density of the overlying blood. Usually after six to 12 months, depending on the extent of obstruction, the hemorrhages and generalized retinal edema gradually disappear and the sequelae of the chronic phase begin with the development of new collateral circulation, cystoid edema of the macula, and hard exudates. The new vessel formations may either occur in the plane of the retina or extend into the vitreous cavity. These new vessels may sometimes rupture with resultant hemorrhage into the vitreous cavity and retina. The visual loss at this stage is caused by involvement of the macula by cystoid edema or hemorrhage from adjacent new vessels into the retina or vitreous. Recovery of visual function, according to Wise, Dollery, and Henkind,[2] is thought to be dependent on the small veins draining the macula.

Photocoagulation in the chronic phase, usually after six months, is useful in preventing new vessel formations associated with bleeding into the vitreous. Hard exudates and edema will disappear.[3-6] In this study, the acute phase is defined as that period during which visual symptoms have been present for not more than 60 days. In the acute phase of branch vein occlusion, there is a progressive venous congestion, retinal edema, and retinal hemorrhage, each compounding the other. Photocoagulation used during the acute phase may impede progression of the congested phase thereby reducing new vessel formation, cystoid edema, and hard exudates that develop in the chronic phase.

Treatment by photocoagulation in the acute phase of retinal branch vein occlusion is an attempt to preserve macular function and to prevent the retinal damage that occurs with the chronic phase. The fovea has a blood supply that is independent of the retinal circulation. According to Gass,[7] the fovea may be involved by edema arising from the adjacent structures.

Two events prevent macular damage in

This is an abridgment of a thesis submitted by Dr. Wetzig as partial fulfillment of requirements for associate membership in the American Ophthalmological Society, May 1978.

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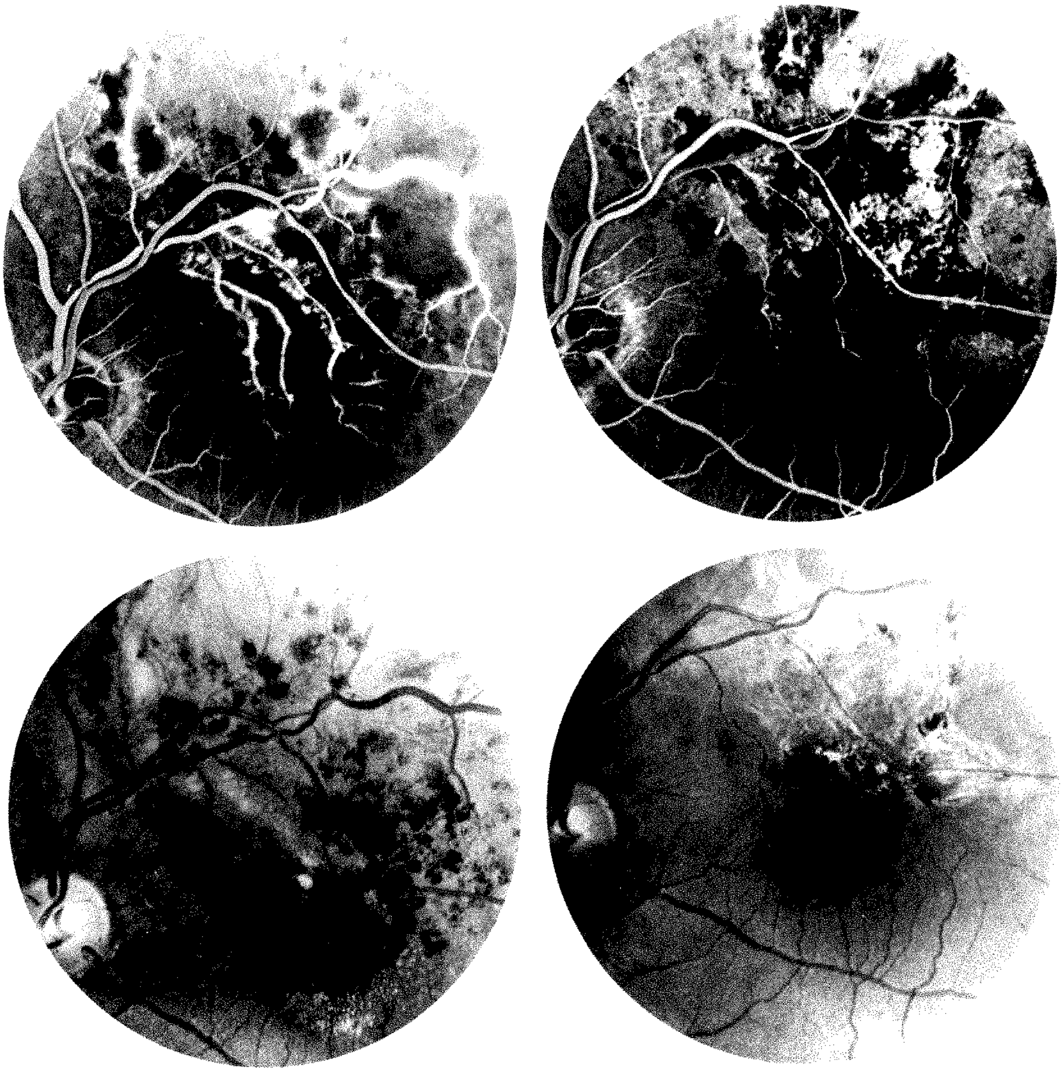

Figure (Wetzig). Top left, Pretreatment 15 second-fluorescein angiogram shows obstruction at the site of the second and third arteriovenous crossing with congestion and dilation of the vein with venous stasis and perivascular fluorescein leakage. Dilation of the capillaries and diffuse leakage with areas of perfusion are seen above the macula at the obstruction site (Patient No. 11). Top right, Four weeks after xenon photocoagulation 15 second-fluorescein angiogram shows restoration of venous circulation in the superior temporal vein with disappearance of congestion and perivenous leakage. There is no sign of diffuse leakage throughout the smaller venules. Bottom left, Pretreatment fundus photograph shows superior temporal branch vein occlusion at the site of the second and third arteriovenous crossing. Diffuse hemorrhages are scattered throughout the involved area, extending into the macula and fovea with fine exudates below the macula. Bottom right, Fundus photograph 17 months after xenon photocoagulation shows no hemorrhages or exudates in the macula. Flame hemorrhages in the superior temporal quadrant have disappeared. Xenon photocoagulation scars can be seen and a preretinal gliosis is seen along the vessels of the involved area.

branch vein occlusion. First, the formation of a scar between the fovea and the adjacent involved area provides a barrier preventing progression of edema and extravasation of blood into the fovea. Second, the massive destruction of the capillary bed reduces the input of the arterial blood supply, allowing the remaining intact capillary bed to drain into the adjacent, less congested, intact capillary bed near the intercapillary communicating vessels. Based on this rationale, a pilot study was done on 28 eyes with temporal branch vein occlusion that were treated by photocoagulation during the acute phase.

SUBJECTS AND METHODS

Twenty-eight patients who complained of visual loss and had occlusion of two or more branches of the central retinal vein were selected for treatment. Only one branch involved the temporal aspect. The onset of the visual symptoms was three to 60 days before treatment, with an average of 39 days. Acute congestion manifested by venostasis, superficial retinal hemorrhages, and generalized retinal edema and cotton-wool exudates in the involved areas were evident. All cases involved the arteriovenous crossing nasal to the macula, and the fovea itself was involved to some degree. Fluorescein angiography showed no evidence of new vessel formation.

This study included a total of 28 eyes (Table 1). The patients varied in age from 41 to 85 years with an average age of 69.6 years, and median age of 71 years. There were 12 men and 16 women. Quadrants involved were the superotemporal, 19; inferotemporal, seven; inferotemporal and inferonasal, one; superotemporal and superonasal, one. The follow-up period ranged from one to eight years, with an average of 3.3 years, and a median of 2.9 years. The concomitant diseases included hypertension in 13; cardiac insufficiency in one; diabetes mellitus in three (no other evidence of diabetic retinopathy in either eye); and generalized arteriosclerosis in two. In all cases, clinical examination before treatment revealed macular hemorrhages and foveal edema.

An initial refraction by the examiner was performed to determine the best corrected visual acuity. All patients were examined in the same standard eye manner, which included a Snellen chart projected with mirrors at a distance of 20 feet. Applanation tonometry, visual fields (in most cases) with the Goldmann perimeter, ophthalmoscopic examination with the indirect ophthalmoscope and the stereoscopic biomicroscope, stereoscopic fundus color photography, and fluorescein angiography were conducted before treatment. Two photocoagulators were used for treatment, the xenon photocoagulators and the argon laser photocoagulator. Each pupil was widely dilated with 0.25% scopolamine and 10% phenylephrine solution. When the xenon photocoagulator was used, diazepam (Valium) was administered slowly intravenously, until the patient had a change in mental sensorium, usually 5 to 10 mg. Following this, 3 ml of 2% lidocaine (Xylocaine) was injected retrobulbarly. Treatment was then directed to the geographic areas involved. Six-degree burns of moderate intensity, sufficient to produce yellow coagulation, were placed in an irregular pattern along the course of the vascular arcades to cover the entire affected area. Three-degree burns of similar intensity were placed between the involved area and the fovea. When indicated, three-degree burns were extended in a crescentic pattern to encompass the temporal aspect. Approximately 40 to 50 such applications were made in the quadrant involved (Figure, top right).

A similar technique was used with the argon laser photocoagulator except that intravenous diazepam and retrobulbar anesthesia were not used. In these cases,

TABLE 1

DATA BEFORE AND AFTER TREATMENT WITH PHOTOCOAGULATION IN 28 CASES OF RETINAL VEIN OCCLUSION*

Patient No., Age (yrs), Sex	Quadrant Involved	Duration (days)	Visual Acuity		Length of Follow-up (mos)	Concomitant Diseases	Clinical Appearance After Treatment
			Initial	Final			
1, 72, M	ST	24	6/24 (28/80+)	6/7.5 (20/25+)	26	Hypertension	Dry
2, 62, F	IT	60	6/15 (20/50)	6/7.5 (20/25+)	53	Hypertension	Dry
3, 68, M	IT	72	6/18 (20/60)	6/60 (20/200−)	82		Dry; cystoid edema in macula
4, 77, F	IT	30	6/9 (20/30)	6/15 (20/50−)	49	Cardiac insufficiency; CVA	Dry
5, 70, F	IN-IT	4	6/12 (20/40)	3/90 (10/300−)	84	Hypertension	New hemorrhage after 4 yrs
6, 83, M	ST	2	6/18 (20/60)	6/15 (20/50)	12		Dry
7, 67, F	ST	5	6/120 (20/400)	6/120 (20/400)	34	Hypertension	Cystoid edema and atrophy in macula
8, 71, F	IT	3	6/60 (20/200)	6/12 (20/40)	14		Dry
9, 76, F	ST	30	6/120 (20/400)	0/000 (10/300)	59	Diabetes and hypertension	Dry
10, 41, M	IT	60	6/15 (20/50)	6/9 (20/30+)	47	Hypertension	Dry
11, 64, F	ST	60	1/90 (3/300)	6/12 (20/40+)	17	Hypertension	Dry
12, 63, M	ST	14	6/18 (20/60)	6/12 (20/40)	12		Dry
13, 67, M	IT	24	6/60 (20/200)	6/60 (20/200−)	31		Cystoid edema in macula
14, 81, F	ST	14	6/9 (20/30)	6/120 (20/400+)	12	Arteriosclerosis	Hemorrhages and edema in macula

TABLE 1 (*Continued*)

DATA BEFORE AND AFTER TREATMENT WITH PHOTOCOAGULATION IN 28 CASES OF RETINAL VEIN OCCLUSION*

Patient No., Age (yrs), Sex	Quadrant Involved	Duration (days)	Visual Acuity		Length of Follow-up (mos)	Concomitant Diseases	Clinical Appearance After Treatment
			Initial	Final			
15, 64, M	ST	60	6/30 (20/100)	6/7.5 (20/25+)	24		Dry; pigmentary change in macula
16, 75, F	ST	3	3/90 (10/300)	6/60 (20/200)	27		Dry
17, 72, F	ST	60	6/60 (20/200)	6/60 (20/200)	54	Hypertension and diabetes	Maculae atrophy
18, 68, M	ST	21	6/120 (20/400)	6/60 (20/200)	68	Hypertension	Dry; pigment deposit in macula
19, 71, M	IT	30	1.5/90 (5/300)	3/90 (10/300)	12		Dry macula
20, 76, F	ST	14	6/90 (20/300)	6/9 (20/30)	40	Hypertension	Dry
21, 73, F	ST	14	6/9 (20/30)	6/21 (20/70)	42		Macula atrophy
22, 72, F	ST	60	6/60 (20/200)	6/60 (20/200)	35	Hypertension and diabetes	Exudates temporal to macula
23, 59, M	ST	14	6/7.5 (20/25)	6/7.5 (20/25)	38	Hypertension	
24, 76, M	ST	60	6/120 (20/400)	6/90 (20/300)	84	Arterio-sclerosis	Dry; macular atrophy
25, 85, F	ST-SN	3	6/90 (20/300)	6/30 (20/100)	96	Retinal detachment	Dry
26, 71, F	ST	30	2/90 (6/300)	6/120 (20/400)	35		Dry; macular atrophy
27, 59, M	ST	14	6/7.5 (20/25)	6/7.5 (20/25)	18		Cystoid edema
28, 66, F	ST	60	6/120 (20/400)	6/7.5 (20/25)	14	Hypertension	Dry

*ST indicates superior temporal; IT, inferior temporal; IN, inferior nasal; SN, superior nasal; CVA, cardiovascular accident.

100 to 150 applications were made with the 100- to 200-microspot size at 3 to 5 mW on the nonpulse setting.

In the parafoveal region the 50-microspot size was used to apply an irregular pattern of burns, avoiding the fovea, but otherwise completely covering the affected area, even extending beyond the horizontal raphe. Usually 50 to 60 burns were made with a 0.2-mV nonpulse setting.

Twenty-two patients were treated with xenon arc only; four patients were treated with only argon photocoagulation, and two patients were treated with both xenon and argon photocoagulation. If there was no evidence of clearing in the foveal region in three to four months, further treatment was considered. Immediately after treatment, the patients did not have any discomfort and they were not aware of any change in visual acuity unless the parafoveal treatment technique was used. These patients were aware of round scotomas, which they described as black spots that eventually turned gray after a few weeks, at which time the patient was no longer aware of them.

Follow-up examination consisted of determining the best visual acuity by using techniques identical to those used for the initial examination. The posttreatment visual acuity used in this study to evaluate the efficacy is that level of vision after the vision was stabilized at two consecutive visits and at least a 30-day interval. Applanation tension was taken on each return visit. The patient was first seen two weeks after treatment, at which time color fundus photography and fluorescein angiography were repeated. The patient was seen again at three months and six months after treatment; each time the color fundus photography was repeated. Thereafter, the patient was seen at six-month intervals. Visual acuity usually stabilized after nine to 12 months. Thereafter, fluorescein angiography and color fundus photography were repeated on a yearly basis. In some cases visual fields were also repeated.

There was usually a marked clearing of the edema and hemorrhage in the treated areas two weeks later, and the previously obscured retinal vessels could be seen. The coagulation marks subsequently began to show signs of pigmentation, and the untreated foveal region cleared more slowly. Fluorescein angiography four weeks after treatment showed restoration of circulation in the previously occluded vessels. No sign of paravenous leakage and reduction in the diffuse leakage was noted (Figure, top right). In most cases, the macular edema and hemorrhage cleared after six months. Visual acuity gradually improved after three months. In some cases, cystoid edema did not resolve and the vision remained static or worsened. There was occlusion of branches other than those in the treated areas, with subsequent deterioration of vision.

Visual field patterns were variable. In the pretreatment visual field, the density of scotoma varied with the density of the obstruction, hemorrhage, and exudates. The posttreatment fields were also extremely variable. The only consistent finding was dense scotoma at the treatment site. When the applications were placed in an irregular pattern, the patient was not aware of the scotoma. Fluorescein angiography did not show new vessel formations in any of the treated areas and in no case was there hemorrhage into the vitreous cavity.

RESULTS

The results of treatment were evaluated on the basis of visual improvement (Tables 1 and 2). Visual improvement was defined as an increase of two lines or more on the Snellen chart. Static was a change of less than two lines, and worsening was a decrease of two lines or more. In a total of 28 eyes, 12 (43%) were

TABLE 2

CHANGE IN VISUAL STATUS OF EYES INCLUDED IN THIS STUDY AFTER COMPLETION OF TREATMENT BY PHOTOCOAGULATION

Change in Visual Status after Treatment	No. of Eyes	Percent
Improved	12	43
Static	11	39
Worse	5	18
Total	28	100

improved, 11 (39%) were static, and five (18%) were worse (Table 3). The cause of worsening in three cases was progression of cystoid edema and in one case progression of the occlusive disease in vessels in other untreated areas of the retina. Those cases that remained unchanged indicated cystoid edema or atrophy in the region of the macula. A case report of patient No. 11 follows.

CASE REPORT

A 64-year-old woman was referred for evaluation of branch vein occlusion. She complained of blurring vision in the left eye for two months. Medical history revealed essential hypertension of several years' duration.

On ophthalmoscopic examination best corrected visual acuity was R.E.: 6/6 (20/20) and L.E.: 1/91 (3/300). Applanation pressure was R.E.: 14 mm Hg and L.E.: 17 mm Hg. The right eye was normal. Ophthalmoscopic examination of the left eye disclosed flame hemorrhages scattered throughout the superior temporal quadrant with hemorrhages extending into the fovea and beneath it. There was marked constriction at the first arteriovenous crossing, the presumed site of the occlusion. Yellow exudates were noted below the macula (Figure, bottom left). Fluorescein angiography revealed obstruction distal to the first arteriovenous crossing with delayed emptying of dye, perivenous leakage, and diffuse leakage into the adjacent structures (Figure, top left).

Xenon photocoagulation with 6-degree aperture was applied along the course of the superior temporal vessels and marks were made immediately above the macula. These marks were more evident in the late follow-up pictures as they were not apparent in the presence of hemorrhages and exudates (Figure, bottom right). Repeat fluorescein angiography four weeks after treatment (Figure, top right) revealed apparent resolution of the obstruction as indicated by the absence of delayed leakage from the superior temporal vein, reduction in caliber of vessels, and the absence of perivenous leakage and diffuse leakage.

At the last follow-up visit 17 months after treatment visual acuity was 6/12 (20/40+). The macula was flat, dry, and uninvolved. Light coagulation scars were seen immediately above the macula. Above the visualized scars no marks could be seen because of the preretinal gliosis (Figure, bottom right).

DISCUSSION

That 43% of eyes with branch vein occlusion improved in visual acuity is impressive and apparently supports the basic concept of protecting the macula from secondary involvement. However, one must compare these results with the natural course of the disease. The only studies to date on the natural course of the disease are those of Michels and Gass,[8] and Gutman and Zegarra[9] (Table 3). In this study I compared the results of the treatment by photocoagulation with the

TABLE 3

FINAL VISUAL ACUITY OF CASES TREATED WITH PHOTOCOAGULATION AND OF UNTREATED CASES

		Untreated Cases	
	Treated Cases (This Study)	Michels and Gass Series[8]	Gutman and Zegarra Series[9]
Average follow-up (yrs)	3.3	3.6	2.07
6/12 (20/40) or better	11 (39%)	23 (53%)	24 (60%)
6/15 (20/50) through 6/30 (20/100)	4 (14%)	12 (28%)	7 (17.5%)
6/60 (20/200) or worse	13 (46%)	8 (19%)	9 (22.5%)
Total	28 (100%)	43 (100%)	40 (100%)
6/15 (20/50) or better	13 (46%)	26 (60%)	26 (65%)
6/7.5 (20/25) or better	6 (21%)	13 (30%)	17 (42.5%)

TABLE 4

INITIAL VISUAL ACUITY OF CASES TREATED WITH PHOTOCOAGULATION AND OF UNTREATED CASES

	Treated Cases (This Study)	Untreated Cases	
		Michels and Gass Series[8]	Gutman and Zegarra Series[9]
6/12 (20/40) or better	6 (21%)	17 (40%)	17 (42.5%)
6/15 (20/50) through 6/30 (20/100)	7 (25%)	13 (31%)	11 (27.5%)
6/60 (20/200) or worse	15 (54%)	12 (29%)	12 (30%)
Unknown		1	
Total	28	43	40
6/15 (20/50) or better	8 (28.5%)	19 (44%)	20 (50%)
6/7.5 (20/25) or better	2 (7%)	3 (7%)	9 (22.5%)

observations of Michels and Gass, and of Gutman and Zegarra (Tables 3-5).[8,9] In the present study there are few patients in the group with better visual acuity, because those with near normal vision were excluded and those with worse visual acuity were specifically considered for this study. The follow-up of the treated cases and the nontreated cases indicated some improvement in visual acuity in both groups. In analyzing the final results of patients with visual acuity of 6/60 (20/200) or less (Table 5), treatment was not effective.

The basic concept of a protective barrier and reduction in overall circulation protecting the fovea from secondary involvement following branch vein occlusion appears not to have influenced the course of the final result as compared to the untreated cases. I believe the use of photocoagulation in the proposed manner is not of obvious major benefit and the findings are inconclusive. Patients were not treated in the same stage of the disease or in an identical manner. If there were massive hemorrhages and exudates in the perifoveal region and underlying structures, caution was taken to avoid coming too close to the fovea. Only when the obstruction was partial and the landmarks could be seen clearly could photocoagulation marks be placed more accurately in an attempt to barricade the fovea from the advancement of blood and edema in the adjacent area. Additionally, the numbers are small, making statistical evaluation difficult and stricter criteria must be imposed to ensure proper evaluation of the natural course of the disease.

More conclusive evaluation can be obtained only when a prospective study on a

TABLE 5

CHANGE IN VISUAL STATUS OF CASES AFTER TREATMENT BY PHOTOCOAGULATION (THIS STUDY) AND UNTREATED CASES WITH INITIAL VISUAL ACUITY OF 6/60 (20/200) OR LESS

Change in Visual Status	Treated Cases (This Study)	Untreated Cases	
		Michels and Gass Series[8]	Gutman and Zegarra Series[9]
Improved	6 (40%)	8 (67%)	6 (50%)
Unimproved or Worse	9 (60%)	4 (33%)	6 (50%)
Total	15 (100%)	12 (100%)	12 (100%)

large number of patients with a more similar pretreatment status, treated under controlled conditions, and evaluated by unbiased observers is made, as in the cooperative study of photocoagulation in the treatment of diabetic retinopathy.[10]

SUMMARY

Twenty-eight eyes with retinal branch vein occlusion were treated by photocoagulation. The clinical posttreatment findings indicated rapid clearing of hemorrhages and exudates. Visual acuity in 12 (43%) eyes improved, in 11 (39%) remained static, and in five (18%) worsened.

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OPHTHALMIC MINIATURE

The mind of a bigot is like the pupil of the eye; the more light you pour upon it, the more it will contract.

Oliver Wendell Holmes

NEONATAL RETINAL HEMORRHAGES AND INFLUENCE OF PERINATAL FACTORS

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Frequency of retinal hemorrhages in the newborn infant has been reported to be between 2.6 and 50%.[1-6] These authors postulate that hemorrhages are caused by fetal factors or maternal factors, or both. Fetal factors include high pressure of the cavernous sinus, increased intracranial pressure, asphyxia, obstetrical trauma, disorders of blood coagulation, vitamin K_1 deficiency, depression of the infant as indicated on the Apgar score, weight and cephalic circumference, sex, cephalic molding, and the stress of the first breathing.[7-13]

Maternal factors include parity, duration of first and second stage of labor, the time between the rupture of the membranes and delivery, umbilical cord around the fetal neck, type of delivery, and the like.

We report herein the investigation of the above factors of labor in the newborn infant that may influence the occurrence of retinal hemorrhage.

SUBJECTS AND METHODS

A group of 234 newborn infants were separated into two groups. The first group (normal) included healthy infants, born at term of mothers without associated disease. Delivery was spontaneous with vertex presentation. None of the mothers in this group took drugs such as aspirin, which might modify the blood coagulation. The second group (abnormal) included all newborn infants depressed during their first minute of life, those with neonatal abnormalities, low birth weight, the product of an abnormal labor, or whose mothers had some associated disease.

The eyes of the neonates were studied by indirect ophthalmoscopy after pupillary dilation with cyclopentholate. The eyelids were held open with a Barraquer blepharostat. Two ophthalmologists made the observations of the infants, and each drew separate retinal figures. The retinal hemorrhages were classified as peripapillary, macular, peripheral, superficial or deep, preretinal, or vitreal.

The Student T-test and Kolmogorov-Smirnov tests were divided according to a population distribution into normal and abnormal groups. Some variables were analyzed by Chi-square and Fisher tests.

RESULTS

One hundred ninety-six infants were delivered vaginally; 38 were delivered by cesarean section. Retinal hemorrhage was

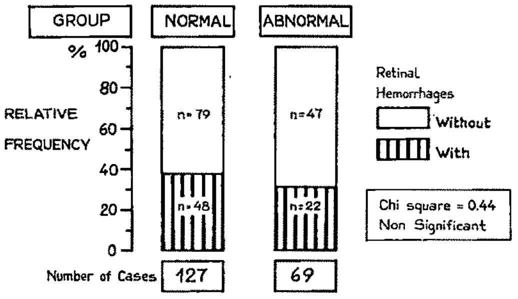

Fig. 1 (Besio and associates). Frequency of retinal hemorrhages in two groups of neonates: NORMAL group, mothers without pathology and normal labor; ABNORMAL group, mothers with pathologic pregnancies or labors (excluding cesarean section).

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AMERICAN JOURNAL OF OPHTHALMOLOGY 87:74-76, 1979

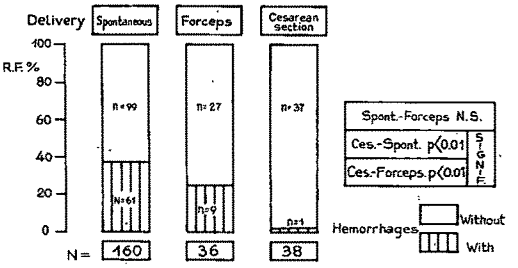

Fig. 2 (Besio and associates). Frequency of retinal hemorrhages of the neonate according to type of delivery.

found in 70 of the 196 vaginally delivered infants and in one infant born by cesarean section. Hemorrhage occurred in 61 (38%) of 160 spontaneous births and in nine (25%) of 36 forceps deliveries.

Of the group classified as normal, retinal hemorrhages were found in 48 (37.8%). In infants classified as having a complicated birth, retinal hemorrhages were found in 22 (31.9%). No statistically significant difference was found in the incidence of retinal hemorrhage between the normal and the abnormal group, and no statistical correlation was found with any of the fetal or maternal factors studied. The only significant difference was between cesarean and vaginal births. No statistical difference in the location of the hemorrhage was evident.

More than 50% of the infants still showed hemorrhage on the seventh day after birth. After 28 days, only one infant had a retinal hemorrhage, which disappeared at age 35 days.

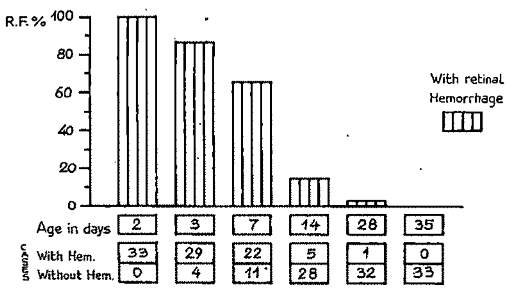

Fig. 3 (Besio and associates). Time of disappearance of retinal hemorrhages in the neonate during the first month of life.

The frequency of retinal hemorrhages in the series agreed with the results found by others.[5,6,14] Vaginal birth significantly increased the production of hemorrhage, and there was a low incidence of hemorrhage by cesarean birth. The mechanism by which vaginal birth produces hemorrhage was not clarified but may be related to the compression exerted on the fetal head through the birth canal. During uterine contractions in the second stage of labor the fetal head was two to four times more compressed than other parts of the fetal body.[15-18]

SUMMARY

We studied fundus oculi in 234 newborns, divided into two groups of mothers and neonates with or without disease. The overall frequency of retinal hemorrhages was 30.3% (71 neonates). In each group, we studied the association between retinal hemorrhage and several variables of labor and the newborn infant. Statistically significant differences were found in the incidence of hemorrhage in one (2.6%) neonate born by cesarean section, as compared with 61 (38%) spontaneous or 9 (25%) forceps delivery.

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OPHTHALMIC MINIATURE

Eyeglasses had been in use since the turn of the century, allowing old people to read more in their later years and greatly extending the scholar's life study. The manufacture of paper as a cheaper and more plentiful material than parchment was beginning to make possible multiple copies and wider distribution of literary works.

Barbara W. Tuchman,
A Distant Mirror—The Calamitous 14th Century
New York, Alfred A. Kopf, 1978

EXACERBATION OF SENILE MACULAR DEGENERATION FOLLOWING CATARACT EXTRACTION

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Cystoid macular edema, the most common cause of decreased visual acuity following cataract extraction, is the cause for permanent visual loss in some patients.[1-4] Less common causes are ischemic optic neuritis,[5] retinal detachment,[6] choroidal detachment, and hypotony with striae. Exacerbation of senile macular degeneration from a nonexudative to an exudative phase following cataract extraction has not received much attention.[7] We report herein on six eyes in four patients that developed retinal pigment epithelial detachments secondary to senile macular degeneration that seemed to be precipitated by cataract extraction.

CASE REPORTS

Case 1—An 86-year-old woman had painless, progressive loss of vision in both eyes for 12 years. A preoperative examination on April 11, 1972, revealed that best corrected visual acuity was 1/120 (3/400) in the right eye and 6/30 (20/100) in the left eye. Slit-lamp examination revealed dense nuclear cataracts in both eyes compatible with her visual acuity. Ophthalmoscopic examination revealed pigment derangement and drusen in the macula of both eyes. There were three small subretinal hemorrhages temporal to the fovea in the right eye, but no subretinal elevation. The patient underwent an uneventful intracapsular cataract extraction of the right eye on April 21, 1972, by cryoextraction using alpha chymotrypsin and a sector iridectomy. Postoperative examination on May 3, 1972, revealed visual acuity had improved to 6/60 (20/200) with a +10 diopter lens. Ophthalmoscopic examination revealed the macula to be flat, but the three small subretinal hemorrhages temporal to the macula were still present. On May 17, 1972, visual acuity remained at 6/60 (20/200) with a +10 diopter lens, and ophthalmoscopic examination revealed fluid beneath the retinal pigment epithelium with several scattered subretinal hemorrhages in the macular area of the right eye (Fig. 1, top left). Fluorescein angiography on Jan. 4, 1973, revealed a 2.5-disk diameter retinal pigment epithelial detachment of the macula with several scattered subretinal hemorrhages (Fig. 1, top right). Argon laser photocoagulation on Feb. 23, 1973, was not beneficial. The patient's visual acuity has remained at 3/120 (10/400) in the right eye since that time. The visual acuity in the left eye became progressively worse. Examination on Feb. 5, 1976, revealed best corrected visual acuity was 6/120 (20/400) in the left eye. Slit-lamp examination revealed dense nuclear sclerosis compatible with visual acuity. Ophthalmoscopic examination of the left eye showed derangement of the retinal pigment epithelium in the macular area and drusen with no subretinal fluid or hemorrhage. Fluorescein angioscopy showed no leakage of fluorescein. The patient underwent an uneventful intracapsular cataract extraction of the left eye on March 3, 1976, by cryoextraction using alpha chymotrypsin and a peripheral iridectomy. She had an uneventful immediate postoperative course. Postoperative examination on March 18, 1976, revealed visual acuity in the left eye was 3/240 (5/400) with a +10 diopter lens. Ophthalmoscopic examination revealed a 1.5 disk diameter retinal pigment epithelial detachment of the macula with hemorrhages along its temporal margin (Fig. 1, bottom left). Fluorescein angiography revealed the retinal pigment epithelial detachment with a subretinal neovascular membrane (Fig. 1, bottom right). Visual acuity has remained best corrected to 6/60 (20/200) in the left eye, and the retinal pigment epithelial detachment has scarred.

Case 2—An 85-year-old man was seen at the Bascom Palmer Institute in December 1963 with a six-month history of painless, progressive loss of vision in the left eye.[8,9] Best corrected visual acuity at that time was R.E.: 6/12 (20/40) and L.E.: 6/60 (20/200). The patient had bilateral posterior subcapsular cataracts. Fundi were normal except for drusen in the posterior poles, and visual acuity worsened to 6/24 (20/80) in the right eye and hand motions in the left eye. In February 1968, he underwent an uneventful intracapsular cataract extraction of the left eye with best corrected visual acuity of 6/9 (20/30).

On July 30, 1968, he underwent an uneventful intracapsular cataract extraction of the right eye and was discharged on Aug. 4, 1968, after an uneventful postoperative course. On Aug. 15, 1968, examination revealed a serous detachment of the retinal pigment epithelium in the macula of the right eye (Fig. 2, top left) and this was confirmed by fluorescein angiography (Fig. 2, top right). On Oct. 23, 1968, the serous detachment had absorbed except inferior nasally, leaving a well-demarcated area of retinal pigment epithelial atrophy with the underlying choroidal vessels appearing sclerotic. A small area of increased pigmentation and fibrovascular tissue remained in

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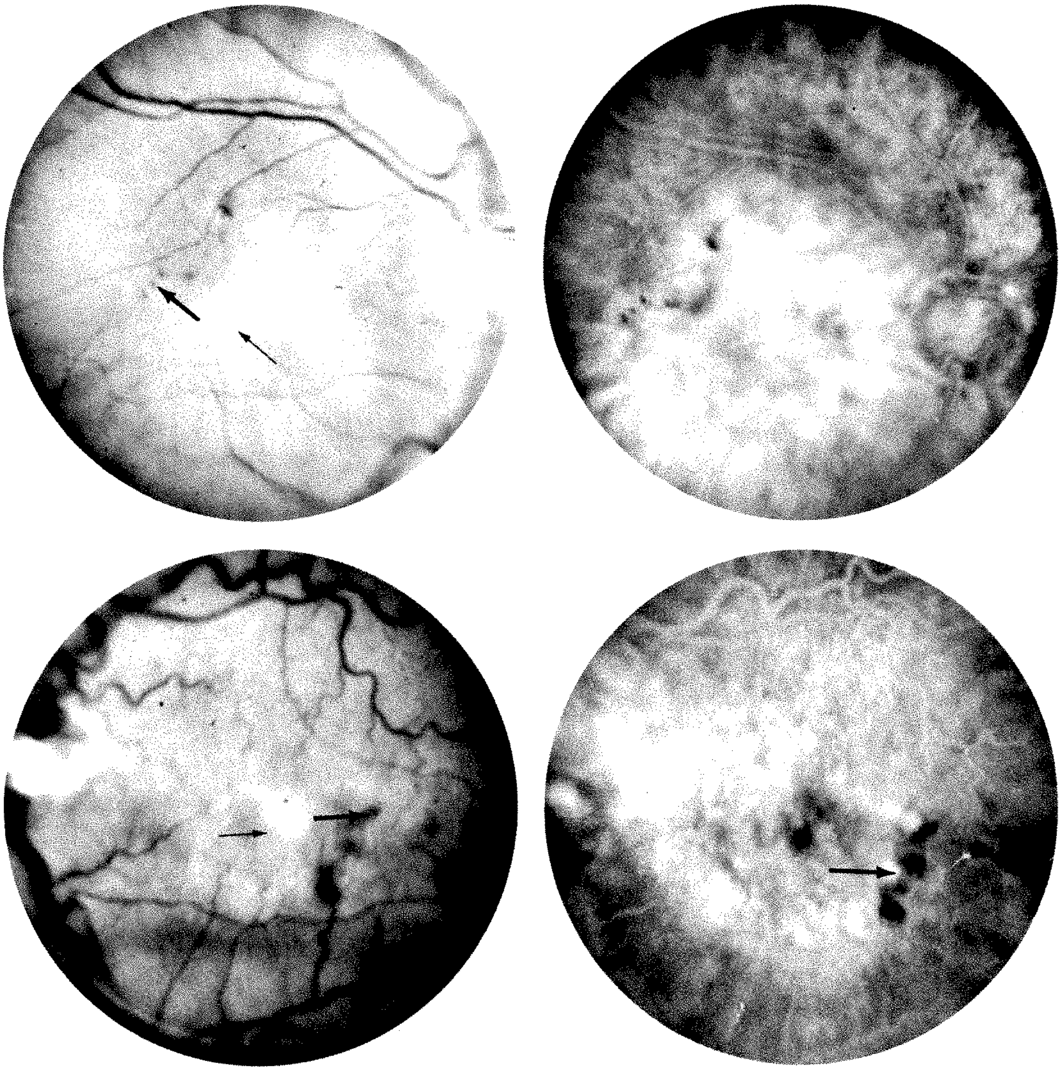

Fig. 1 (Blair and Ferguson). Case 1. Top left, Appearance of macula of right eye on May 17, 1972, with serous detachment of the retinal pigment epithelium (small arrow). Note the presence of the five subretinal hemorrhages (large arrow). Top right, Fluorescein angiography (late phase) demonstrating fluorescein in pigment epithelial detachment and blockage by the subretinal hemorrhage. Bottom left, Appearance of the left macula on Feb. 18, 1976, with serous fluid beneath the retinal pigment epithelium (small arrow) and hemorrhages temporally (large arrow). Bottom right, Fluorescein angiography of the left eye showing neovascular membrane and hemorrhages (arrow).

the inferior nasal aspect of this lesion (Fig. 2, bottom left). This subsequently developed hemorrhages along the nasal margin (Fig. 2, bottom right). Visual acuity has been best corrected to 6/60 (20/200) in the right eye.

Case 3—A 79-year-old man had noticed painless, progressive loss of vision in both eyes since 1970. On June 26, 1973, visual acuity was best corrected to R.E.: 6/60 (20/200) and L.E.: 6/24 (20/80). Moderately dense nuclear cataracts were present in both eyes, compatible with the visual acuity. Ophthalmoscopic examination revealed peripapillary atrophy of the retinal pigment epithelium in both eyes. The macula was normal with no fluid. On Aug. 8, 1973, the patient underwent an uneventful intracapsular cataract extraction of the right eye by means of the cryoprobe with a peripheral iridectomy and alpha chymotrypsin. He developed prostatic obstruction

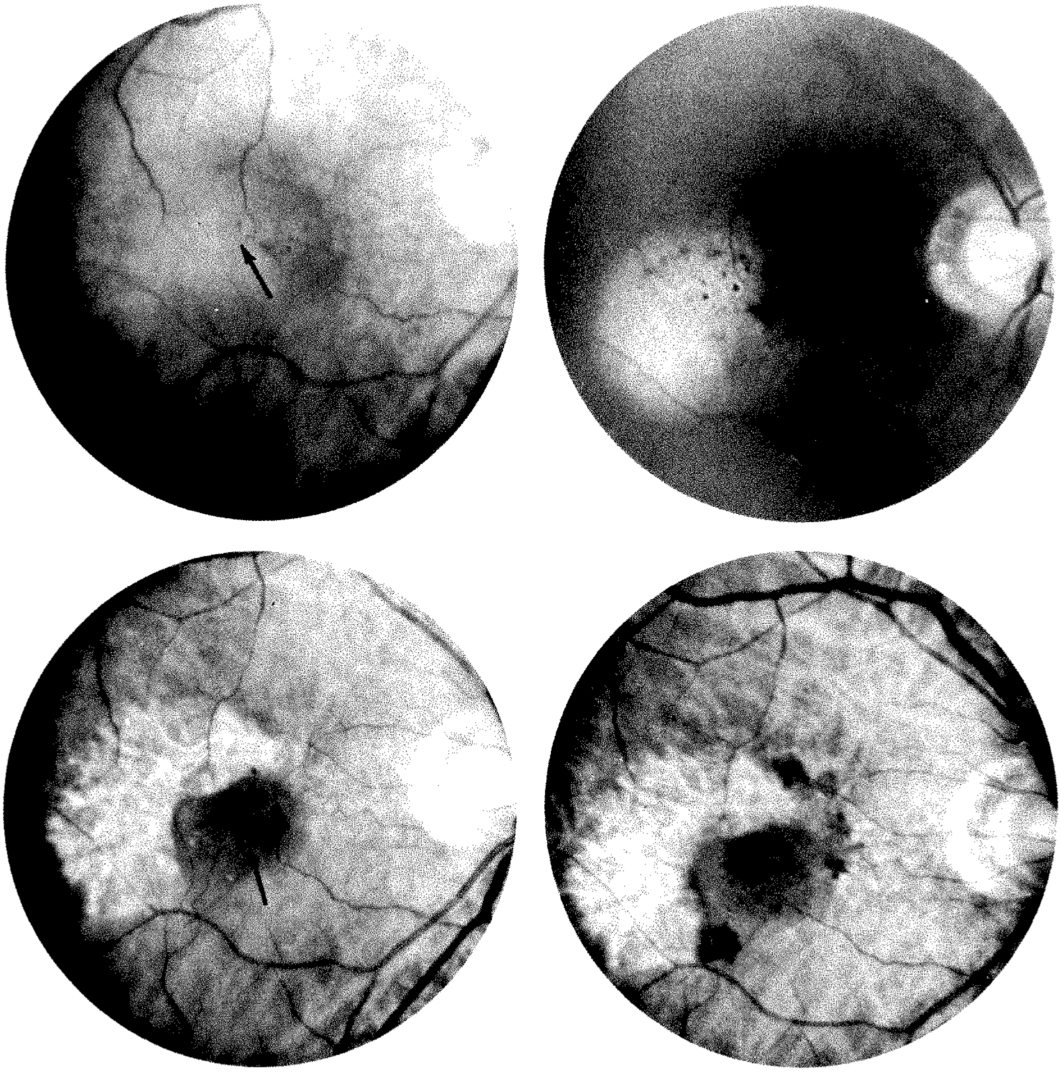

Fig. 2 (Blair and Ferguson). Case 2. Top left, Appearance of the macula of the right eye on Aug. 15, 1968, with serous detachment of retinal pigment epithelium (arrow). Top right, Fluorescein angiography (late phase) demonstrates fluorescein in pigment epithelial detachment. Bottom left, Appearance of macula on Oct. 23, 1968, after most of the serous fluid had been absorbed. Note that the pigment epithelium has atrophied with the underlying visible sclerotic-looking vessels. Inferior nasally is an area of pigment deposition (arrow). Bottom right, Appearance of the macula after subretinal hemorrhages had developed at the nasal margin.

in the immediate postoperative phase and underwent a transurethral resection. Ophthalmoscopic examination on Aug. 27, 1973, revealed the fundus to be normal. On Sept. 1, 1973, examination revealed a hemorrhagic detachment of the retinal pigment epithelium of the right eye (Fig. 3, top left). Fluorescein angiography on June 16, 1974, revealed a hemorrhagic detachment of the retinal pigment epithelium with a subretinal neovascular membrane in the macular area of the right eye (Fig. 3, top right). There was some peripapillary atrophy and hyperplasia of the retinal pigment epithelium around the disk. The retinal pigment epithelial detachment remained essentially unchanged when the patient was last seen on March 14, 1975, and visual acuity was best corrected to 1/20 (4/400).

Case 4—An 81-year-old man came here in June 1973 with bilateral nuclear sclerosis. Preoperative

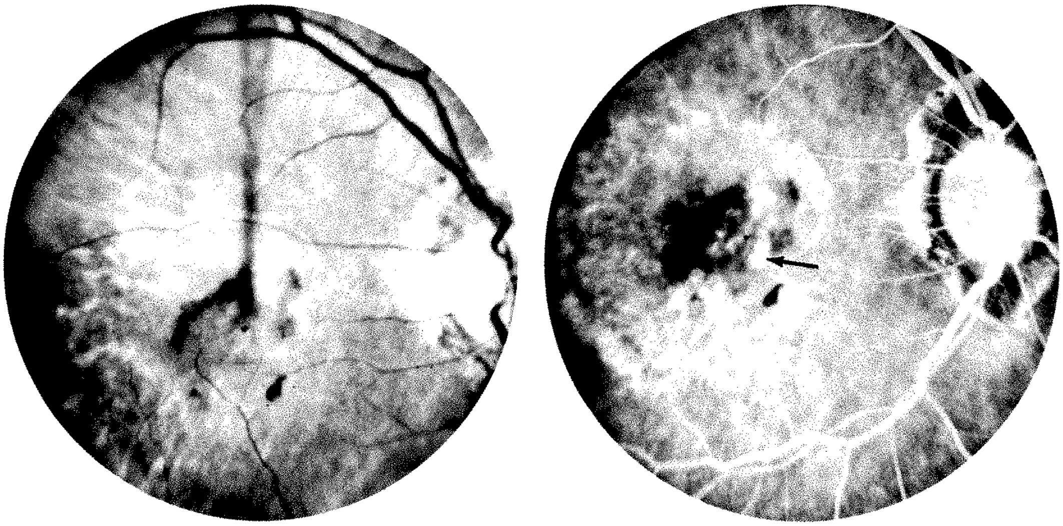

Fig. 3 (Blair and Ferguson). Case 3. Left, Right eye on Sept. 1, 1973, demonstrating hemorrhagic detachment of the retinal pigment epithelium. Right, Fluorescein angiography on June 16, 1974, demonstrating hemorrhagic detachment of the retinal pigment epithelium and subretinal neovascular membrane (arrow).

examination in January 1974 revealed best corrected visual acuity was 6/30−1 (20/100-1) in the right eye and counting fingers at one foot in the left eye. Ophthalmoscopic examination of the right eye revealed derangement of the retinal pigment epithelium. The left fundus was not clear because of the opaque media. The patient underwent an uneventful intracapsular cataract extraction of the left eye on Feb. 1, 1974, by means of the cryoprobe with a peripheral iridectomy. Postoperative examination on Feb. 12, 1974, revealed visual acuity in the left eye had improved to 6/24 (20/80) with correction, and ophthalmoscopic examination revealed pigment mottling in the macular area, with no subretinal fluid. Examination on July 10, 1974, revealed best corrected visual acuity was R.E.: 6/30−1 (20/100-1) and L.E.: 6/18 (20/60). Ophthalmoscopic examination revealed derangement of the retinal pigment epithelium in the macula of both eyes. On July 26, 1974, the patient underwent an uneventful intracapsular cataract extraction of the right eye which was complicated in the immediate postoperative period by a superior temporal choroidal detachment that resolved spontaneously. Examination on Sept. 27, 1974, revealed best corrected visual acuity in the right eye was 6/30 (20/100) with questionable serous elevation of the retinal pigment epithelium in the macula. On Oct. 22, 1974, the pigment epithelial detachment had become much larger and hemorrhagic (Fig. 4, top left). On Dec. 24, 1974, a small hemorrhagic detachment of the retinal pigment epithelium of the left eye was noted (Fig. 4, top right). Fluorescein angiography confirmed the presence of a large hemorrhagic retinal pigment epithelial detachment of the right eye (Fig. 4, bottom left), and a neovascular membrane beneath the retinal pigment epithelial detachment of the left eye (Fig. 4, bottom right). The patient underwent argon laser photocoagulation to the left eye, with flattening of the retinal pigment epithelial detachment. On June 30, 1976, the ophthalmoscopic examination showed a hemorrhagic detachment of the retinal pigment epithelium in the macula of the right eye and a macular scar in the left eye. Best corrected visual acuity at that time was R.E.: 6/120 (10/200) and L.E.: 6/60 (20/200).

DISCUSSION

Gass[8] reported a case of retinal pigment epithelial detachment following cataract extraction. Blair[9] included the same case in a later report (Case 2 in the present report). We described six eyes in four patients who developed retinal pigment epithelial detachments following cataract extraction. All of the retinal pigment epithelial detachments were hemorrhagic. Preoperatively, all patients demonstrated evidence of senile macular degeneration with changes in the retinal pigment epithelium or drusen, or both, on ophthalmoscopic examination. One patient (Case 1) underwent fluorescein angioscopy preoperatively without any evidence of subretinal leakage. This patient had sev-

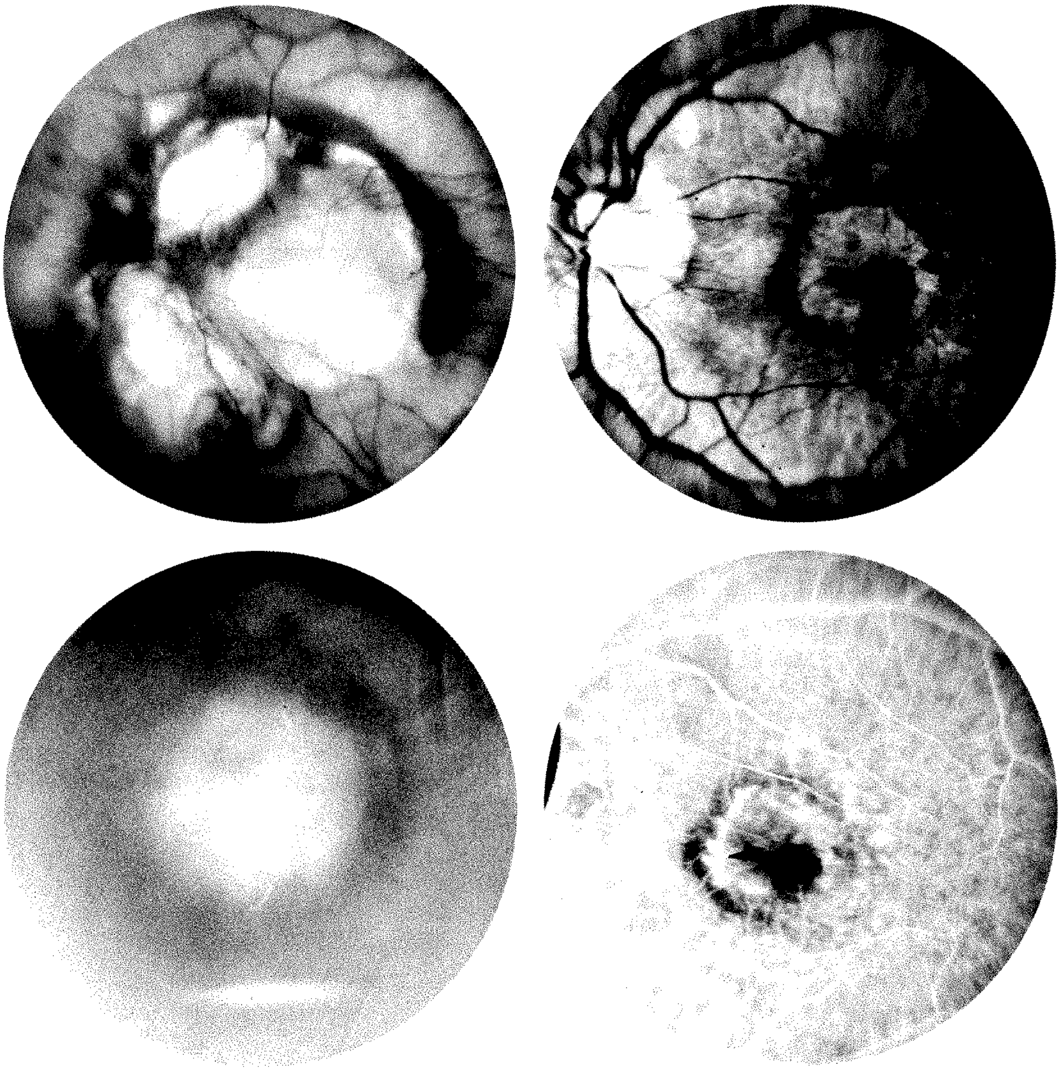

Fig. 4 (Blair and Ferguson). Case 4. Top left, Appearance of macula of the right eye on Oct. 22, 1974, showing the large hemorrhagic detachment of the retinal pigment epithelium. Top right, Appearance of the macula of the left eye on Dec. 24, 1974, showing the hemorrhagic detachment of the retinal pigment epithelium. Bottom left, Fluorescein angiography (late phase) of the macula of the right eye showing fluorescein in the pigment epithelial detachment. Bottom right, Fluorescein angiography (early phase) of the macula of the left eye showing the prominent neovascular membrane (arrow) beneath the retinal pigment epithelial detachment.

eral small subretinal hemorrhages preoperatively without subretinal fluid. Postoperatively, all of the eyes showed evidence of a hemorrhagic detachment of the retinal pigment epithelium manifested by a subretinal hemorrhage on ophthalmoscopic examination or evidence of subretinal neovascular membrane with hemorrhage, or both, on fluorescein angiography.

The period of time between the cataract extraction and the first observation of the retinal pigment epithelial detachment varied from 15 days to 153 days (Table).

TABLE

SUMMARY OF CLINICAL DATA

Case No.	Age (yrs)	Sex	Eye Involved	Onset from Surgery (days)	Type of Retinal Pigment Epithelial Detachment	Final Visual Acuity
1	86	F	R.E.	26	Hemorrhagic	3/120 (10/400)
			L.E.	15	Hemorrhagic	6/60 (20/200)
2	85	M	R.E.	16	Hemorrhagic	6/60 (20/200)
3	79	M	R.E.	25	Hemorrhagic	1/120 (4/400)
4	81	M	R.E.	61	Hemorrhagic	6/120 (10/200)
			L.E.	153	Hemorrhagic	6/60 (20/200)

In four eyes of three patients, the time ranged from 15 days to 26 days. In all cases, there was no daily monitoring of the fundus, so that it is possible that the fundus changes occurred before the time they were first observed. The age at onset ranged from 79 to 86 years, with a mean of 83 years. The average age of 1,158 consecutive cataract patients at the Richmond Eye Hospital in 1975 was 69 years. The patients in our study were on the average 14 years older than the average cataract extraction patient, and this may indicate that the ocular-vascular system of these elderly patients is more prone to this complication.

Because both senile macular degeneration and senile cataract are common causes of decreased visual acuity in the elderly, it might be argued that the findings were coincidental and would have occurred regardless of whether the cataract extraction had been performed or not. Many cases of disciform senile macular degeneration observed in the postoperative phase are present preoperatively; thus, we are not inferring that the cataract extraction frequently precipitates this event. No statistical data are available to determine the frequency of occurrence, and a well controlled study will be needed to determine the true incidence. It may also be argued that in our cases, the retinal pigment epithelial detachment was present before the cataract extraction and was obscured by poor visibility. However, we believe the fundus was adequately visualized to evaluate macular disease preoperatively in most cases, and postoperatively in all cases. Fluorescein angioscopy was negative preoperatively in Case 1; in Cases 1 and 4 visual acuity initially improved after the operation but it deteriorated after the development of the retinal pigment epithelial detachment, and the detachment appeared fresh with red blood or serous fluid, or both, to become cicatricial later in some cases. The temporal relationship in these cases of cataract extraction to the onset of the retinal pigment epithelial detachment, and the involvement of the second eye in a similar manner after cataract extraction in two cases, supports the contention that the cataract extraction in some way precipitated the course of events that led to the hemorrhagic retinal pigment epithelial detachment. In senile macular degeneration, neovascular membranes from the choroid often grow under the retinal pigment epithelium.[10] It could be theorized that the hypotony associated with cataract extraction induces pre-existing subretinal new blood vessels to bleed, leak, or proliferate, causing hemorrhagic retinal pigment epithelial detachments. This seems to be the most plausible explanation. In Case 1, pre-existing small subretinal hemorrhages without subretinal fluid were noted, both in the preoperative and immediate postoperative phases, before the retinal pigment epithelial detachment developed. However, it might be argued that cataract extraction sets up the hemodynamics for the formation of new blood vessels from the choroid in a predisposed eye. Whatever the pathogenesis, one should be particularly cautious in patients who have had cataract extraction followed by exudative senile macular degeneration in one eye and surgery is contemplated in the other.

SUMMARY

Six eyes in four patients had hemorrhagic detachment of the retinal pigment epithelium following cataract extraction. All eyes had evidence of nonexudative senile macular degeneration preoperatively as manifested by drusen or alterations in the retinal pigment epithelium, or both. Postoperatively, all eyes had evidence of a subretinal neovascular membrane manifested by the presence of subretinal hemorrhage or by evidence on fluorescein angiography. We believe cataract extraction may cause changes in the choroidal hemodynamics that allow pre-existing new blood vessels to leak or bleed, or induces the formation of choroidal neovascularization in a predisposed eye.

ACKNOWLEDGMENT

J. Donald M. Gass, M.D., provided records of Case 2.

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THE USE OF STRONTIUM-90 IN THE TREATMENT OF CARCINOMA IN SITU OF THE CONJUNCTIVA

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Intraepithelial or in situ epidermoid carcinoma of the conjunctiva is an uncommon tumor. It remains noninvasive for some time but eventually breaks through the basement membrane.[1] Metastases, mainly to draining lymph nodes, have been reported.[2] The tumor usually appears as an elevated gelatinous plaque and is seldom raised more than 1 to 2 mm above the conjunctival surface. Irvine[3] surveyed a large number of patients by questionnaire. He found that in 65 of 104 (63%) the margin of the tumor was clearly defined and in many of the remaining tumors a 2-mm margin of treatment around the lesion was adequate to encompass it. Of the lesions, 15% were multicentric. The tumor usually involves the corneoscleral limbus and tends to grow over the cornea. If untreated, it may cause severe visual loss and local pain. The confined nature of the tumor, lack of penetration of the basement membrane, and low elevation from the surface of the conjunctiva make them suitable for treatment with strontium-90, with or without prior surgical removal.

Strontium-90 is a pure beta-emitting isotope. It decays to yttrium-90 with a half-life of 28.1 years and with the emission of a beta particle of maximum energy of 0.546 mega electronvolts (MEV). Yttrium-90 decays to stable zirconium-90 with emission of a 2.273-MEV beta particle. In the present series, commercially available ophthalmic applicators were used for treatment; in particular the SIA-1 and SIA-6 applicators (Amersham-Searle). The depth dose characteristics (Table) of these applicators are higher than those reported by other investigators.[4,5] The treatment technique used has been previously described.[6]

CASE REPORTS

Case 1—A 69-year-old man was reported to have a lesion of approximately 4 mm in diameter at the right nasal corneoscleral limbus. Biopsy revealed squamous cell carcinoma in situ. In 1958, the lesion was removed. However, in 1965 the lesion recurred in the same area and was again removed. In 1972, a further recurrence in the area was confirmed by biopsy to be in situ disease. The lesion was removed and strontium-90 was administered from March 6 to April 10, 1972. A surface dose of 750 rads was administered weekly for six weeks to a total dose of 4,500 rads by using an SIA-6 applicator. In 1978, the patient was seen for follow-up and there was no evidence of recurrence or lens opacities.

Case 2—A 66-year-old man had a squamous cell carcinoma in situ removed involving the corneoscleral limbus of the right eye in August 1969. The lesion measured 0.7 × 0.4 cm and was located on the inferior and temporal margins of the corneoscleral limbus and bulbar conjunctiva. Pathologically, there were dysplastic changes at the margin of excision. In December 1969 there was a definite area of recurrence at the inferior aspect of the cornea. From May 12 to June 16, 1970, the patient had six weekly applications of strontium-90 (SIA-6), 750 rads being administered per application to a total dose of 4,500 rads. In 1973 the area had completely healed. Some growth of capillaries in the conjunctiva in the area of the strontium-90 application occurred. A small peripheral cataract was noted. The patient was last seen on March 30, 1978, with no evidence of recurrent tumor and good vision in the

TABLE
OPHTHALMIC APPLICATORS

Depth (mm)	Percentage Depth Dose
0	100
1	64
2	40
3	23
4	13

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right eye. The small peripheral cataract was still present.

Case 3—An 80-year-old man had an eight-month history of irritation of the right eye. Examination revealed a raised 0.5 cm diameter, plaque-like lesion on the temporal surface of the right conjunctiva adjacent to the corneoscleral limbus. He had cataracts in both eyes. Visual acuity in the left eye was 6/24 (20/80) and counting fingers in the right eye. Biopsy revealed dysplasia with focal epidermoid carcinoma in situ. From Dec. 28, 1973, to Feb. 7, 1974, the patient received 7,000 rads of beta irradiation with strontium-90 (SIA-1). He received seven applications at weekly intervals of 1,000 rads each. After treatment, there was erythema in the area treated. There was first degree erythema one month later. He had complete healing of the area with no evidence of recurrence three years later, and 52 months later there was no evidence of disease. Vision in the right eye was unchanged.

Case 4—A 48-year-old man was uniocular and reported to have a large lesion of the left conjunctiva with extension onto the cornea and around the conjunctiva for a distance of 9 mm. The patient was treated in 1951 and was given both superficial x-rays and strontium-90.

In September 1958, a recurrence of the tumor was noted with the lesion extending over the nasal bulbar conjunctiva from plica semilunaris to cornea and extending 2 mm onto the cornea. The lesion was surgically removed. In June 1960, a large central posterior polar cataract was noted and removed. There were further recurrences of disease in the cornea in 1962, 1963, 1966, and 1967, which were treated surgically. A further recurrence in March 1968 was treated by superficial x-ray by using a 2-mm diameter field and administering 4,500 rads. In March 1978 there was no recurrence of the lesion and the patient's visual acuity was 6/60 (20/200).

This case represents a different therapeutic approach in that superficial x-rays were used in addition to beta irradiation. It demonstrates that retreatment with x-rays is possible with tumor control and retention of vision.

DISCUSSION

Recurrence rates after surgical removal of squamous cell carcinoma in situ of the conjunctiva vary between 12 of 59 (20%)[3] and 8 of 22 (36%). This figure may be underestimated because in many of the cases reported, the follow-up period is short, and lesions may occur at long periods after initial treatment.[7] In the present series, two of the three patients who developed recurrence of tumors did so seven years after initial therapy. After a first recurrence, there is a tendency for further recurrences.[8]

McDonald[9] and Carroll[10] reported a total of nine cases of squamous cell carcinoma in situ, treated with beta irradiation with no recurrences. McDonald used a mean dose of 10,000 reps (roentgen equivalent physical), but he reported that some of the cases were cured with less than 5,000 reps. Murphy[11] treated patients with squamous cell carcinoma in situ with strontium-90 to 20,000 rads (surface dose) with no complications. Some authors have reported a high incidence of cataract formation.[4,12,13] However, the dosage used in these series was high. Lommatzsch[14] reported that in two of 15 cases glaucoma developed as a complication of beta radiation using doses of 12,000 to 18,000 reps. In two of our cases (Cases 1 and 2), and in McDonald's and Wilson's case,[9] a lower dose of beta radiation was adequate to control the tumor. In our series with surgical removal or small recurrence, 4,500 rads administered in six weekly fractions of 750 rads was sufficient to render the patients disease-free. One of our patients (Case 3) was treated without prior surgical removal and control was obtained with 7,000 rads administered at 1,000 rads weekly.

A surface dose of 4,500 to 5,000 rads represents a dose of 900 to 1,000 rads to the lens. This dose administered over six to seven weeks, rarely causes lens opacity.[12]

The patient described in Case 2 did develop a small peripheral cataract with this dose but no visual impairment resulted. Doses of 7,000 rads at the surface of the corneoscleral limbus do cause cataracts in a higher proportion of patients, but seldom are these progressive, nor do they lead to significant loss of vision.

We have been unable to obtain any details of the initial radiation treatment in Case 4. The tumor has been controlled for ten years with superficial x-rays (4,500 rads).

In the small percentage of patients with multifocal tumors, treatment to the entire conjunctiva may be appropriate. Lederman[15] described a technique for beta irradiation of the whole conjunctiva. We do not routinely advocate treatment to the entire conjunctiva, as suggested by Lederman.

SUMMARY

Four patients with carcinoma in situ were treated with strontium-90 beta ray application. The dose used was 4,500 rads after surgical removal or with small recurrent tumor and 7,000 rads for primary treatment. All patients were disease-free at follow-up periods of 48 to 320 months after treatment. One patient required superficial x-ray treatment for recurrent disease. No serious complications developed with the treatment and no progressive cataracts have occurred.

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MONOCULAR VERTICAL NYSTAGMUS AS AN INITIAL SIGN OF CHIASMAL GLIOMA

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Acquired monocular nystagmus, either horizontal or vertical, is uncommon. Causes of the disorder include multiple sclerosis, brain stem disorders,[1] unilateral amblyopia,[2] spasmus nutans,[3] and possibly cerebral trauma.[4] We report herein a case of a child with monocular vertical nystagmus as the initial sign of chiasmal glioma. Because his eye movement abnormality was first confused with spasmus nutans, correct diagnosis was delayed. Chiasmal mass lesions may produce eye movements that mimic a benign ocular dyskinesia, spasmus nutans.

CASE REPORT

A 5-year-old boy was referred to us on Feb. 14, 1977, for evaluation of monocular vertical nystagmus in the left eye. The parents first noted a vertical jiggling of the patient's left eye at age 3 years. One year later, ophthalmologic examination revealed grossly normal visual acuities bilaterally, but poor patient cooperation prevented precise measurements. Examination and history did not indicate any obvious visual impairment, and a diagnosis of spasmus nutans was made.

In December 1976, the child was examined at a local college of optometry and visual training exercises were prescribed. The mother returned to her primary ophthalmologist for another opinion. He found optic atrophy in the left eye and a left afferent pupillary defect and referred the boy to us for further assessment.

Examination revealed visual acuity of 6/12 (20/40) in the right eye, and questionable light perception in the left eye with a left afferent pupillary defect. Neither proptosis nor limitation of ocular motility was noted. A rapid, small amplitude, vertical, pendular nystagmus in the left eye was observed, that was unchanged with horizontal or vertical gaze. Biomicroscopy indicated a micro-amplitude, rapid vertical nystagmus in the right eye. Even with cinematography, the fine movements of the right eye were not observable. Without ocular motor tracings, conjugacy could not be determined. The right fundus appeared normal, whereas the left showed atrophy of nerve fibers prominent in the papillomacular bundle. General physical examination indicated small stature (third percentile).

Skull films and sella tomography (Fig. 1) showed marked anterior erosion of the sella turcica with undercutting of the anterior clinoids and slight straightening of the dorsum sellae. The left optic foramen was grossly enlarged (Fig. 2). Computed tomography (Fig. 3) revealed an enhanced mass in the region of the optic chiasm with undercutting of the tuberculum. Ventricular enlargement was not present.

We diagnosed chiasmal glioma. Periodic follow-up examinations of the patient revealed no change in his condition, except for mild nerve fiber dropout in the right eye, until March 1978, when his central visual acuity dropped to 6/21 (20/70) in the right eye. Radiation therapy (5,000 rads) was administered, and the patient's condition remains unchanged at the present time.

DISCUSSION

The most common cause of acquired horizontal monocular nystagmus in infants is spasmus nutans,[5] which is characterized by nystagmus, head nodding, and

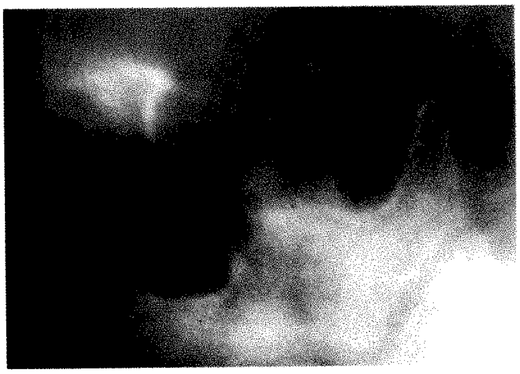

Fig. 1 (Schulman, Shults, and Jones). Lateral tomogram of sella turcica. Note undercutting of the anterior clinoids, "J"-shaped sella, and slight straightening of the dorsum.

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AMERICAN JOURNAL OF OPHTHALMOLOGY 87:87-90, 1979

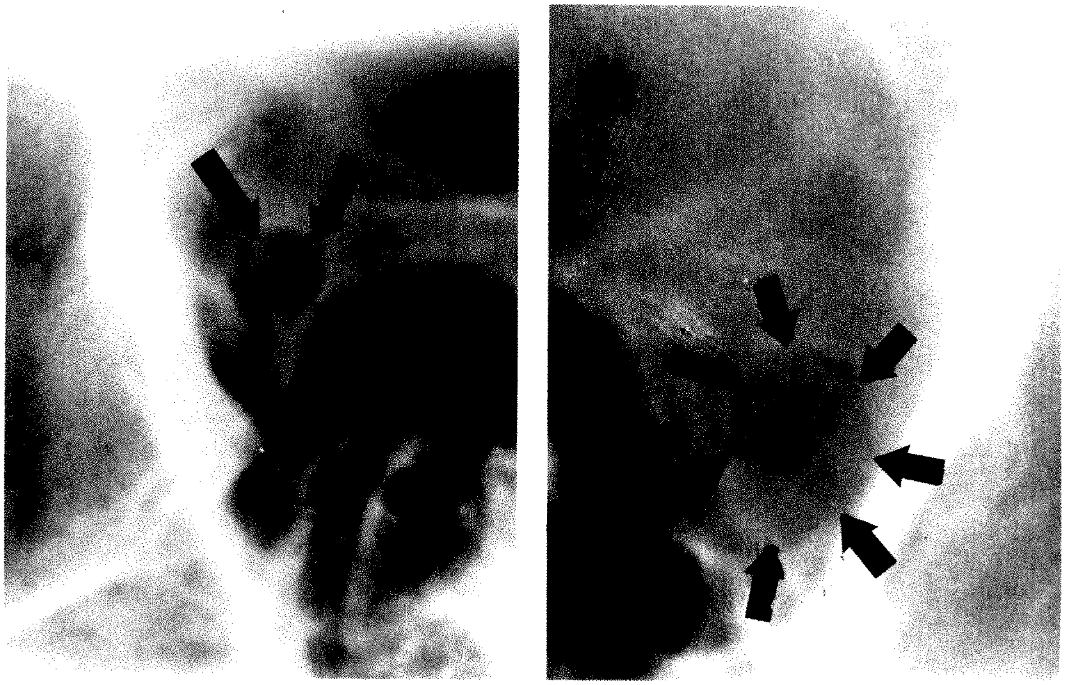

Fig. 2 (Schulman, Shults, and Jones). Optic canal tomograms. Left, The left canal is smoothly enlarged (arrows). Right, The right canal is normal (arrows).

torticollis.[3] Nystagmus in spasmus nutans may be unilateral or bilateral and may occur with or without head nodding or torticollis.[6] It can begin in children as old as 3½ years of age and may last up to 27 months.[3]

Donin[7] reported the occurrence of acquired monocular nystagmus as a symptom of chiasmal glioma in two children. He emphasized the importance of carefully assessing the visual system in such patients before assigning them the diagnosis of spasmus nutans. His first case, a 3-year-old boy with a five-month history of monocular quivering eye movements, subsequently was found to have an anterior visual pathway glioma. Tumor-induced blindness was the cause for his nystagmus. His second case, an 8½-month-old boy, had monocular rapid, horizontal, pendular nystagmus in his left eye. Seven months after it appeared, this ocular movement ceased abruptly and permanently after a fall. Within seven months of the onset of nystagmus, the development of nausea, vomiting and hemiparesis led to examination and subsequent craniotomy, which revealed a large third ventricular astrocytoma.

Udvarhelyi, Khodadoust, and Walsh,[8] in a series of eight children with gliomas of the optic nerve and tract, described a 6-year-old boy with monocular nystagmus preceded by two years of decreased vision in the affected eye. Kelly[9] described a patient whose acquired monocular nystagmus preceded anterior visual pathway glioma. This child was initially believed to have spasmus nutans because of a head nod. Three years later, visual loss prompted a neurodiagnostic evaluation and led to the discovery of optic nerve and chiasm glioma with hypothalmic involvement. A patient described by Walsh and Hoyt[10] had seesaw nystagmus and a chiasmal glioma. When first examined, his hypertrophic left eye exhibited spontaneous vertical movements, while

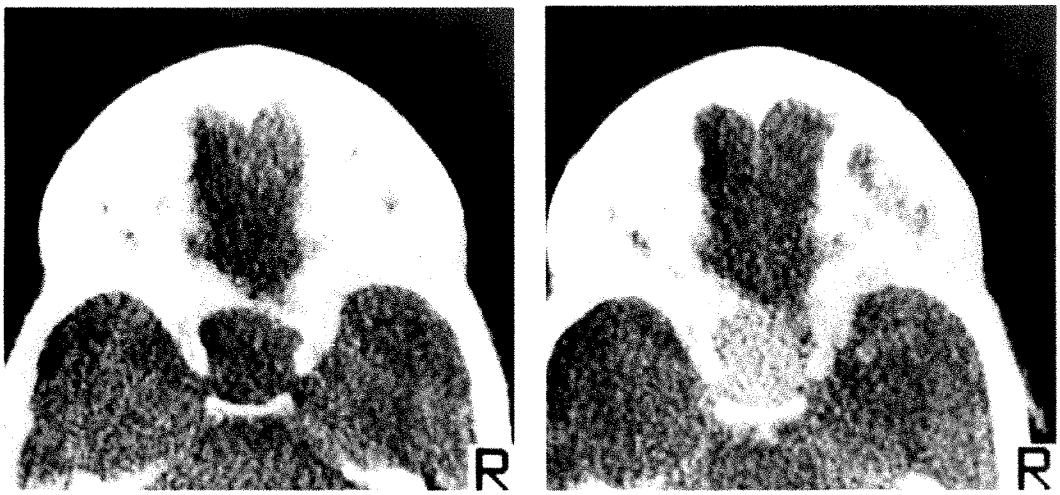

Fig. 3 (Schulman, Shults, and Jones). Computed tomographic scans. Left, Contrast unenhanced. Note marked anterior erosion of tuberculum sellae. Right, Contrast enhanced. A large enhanced mass is evident with hypothalmic extension.

questionable intermittent vertical movements were seen in his right eye. When examined six months later, the boy had seesaw nystagmus.

Though in his earlier writings Cogan[5] states that spasmus nutans may be the only cause of acquired horizontal monocular nystagmus in infancy, he cautions in a subsequent work[11] that monocular vertical nystagmus may indicate chiasmal glioma. Our case substantiates this.

Monocular nystagmus is an uncommon indication of chiasmal glioma. Dodge and associates[12] reported a series of 46 patients with anterior visual pathway gliomas. Of 12 patients whose glioma was confined to the optic nerve alone, none had nystagmus, whereas three of 34 patients with gliomas of chiasm and optic tract had nystagmus. Chutorian and associates[13] described a series of 56 children with optic gliomas. Nystagmus was present in 14 patients at the time of their initial examination. Lloyd's series[14] of childhood gliomas of the optic nerve and chiasm consisted of 41 patients divided into three subgroups. In Group 1, nine patients with solitary optic nerve tumors, nystagmus was not evident. In Group 2, 12 patients with chiasm or bilateral optic nerve involvement, or both, and with a stable course, one blind patient exhibited nystagmus. In Group 3, of 20 patients with extensive nerve chiasm involvement and deteriorating course, four patients had nystagmus. In each of these series a relatively small number of patients had nystagmus as a prominent initial symptom.

SUMMARY

A 5-year-old boy with monocular vertical nystagmus, initially believed to have spasmus nutans, subsequently developed optic atrophy and visual loss. Neuroradiologic investigation indicated probable chiasmal glioma.

The case of this patient re-emphasizes the necessity of careful clinical and radiologic assessment before assuming acquired monocular nystagmus to be a benign and self-limited disorder.

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OPHTHALMIC MINIATURE

Madness is possibly the only disease for which there are no diagnostic tests or laboratory findings to bolster clinical judgment. Perhaps this is why social factors hold sway with reference to the fate of madmen rather than purely medical ones.

Nowadays, . . . I always look a madman straight in the eye at least once—with an ophthalmoscope.

British Medical Journal, 10 June 1978

OPTICOCILIARY VEINS IN A PRIMARY OPTIC NERVE SHEATH MENINGIOMA

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Meningioma originating in the orbital portion of the optic nerve is an uncommon but significant cause of proptosis and visual loss. Most optic nerve sheath meningiomas secondarily involve the orbit by extension from an intracranial site, most often the sphenoid bone.[1,2] Meningiomas can arise primarily from arachnoidal cells of the optic nerve sheath and from ectopic arachnoidal cells within the orbit, but separate from the optic nerve sheath.[2] Opticociliary shunt vessels associated with visual loss and pallor of the optic disk are suggestive of primary or secondary meningioma of the optic nerve sheath.[3,4] Elschnig[5] described such vessels histopathologically in a patient with an optic nerve sheath meningioma in 1898. More recently Rodrigues, Savino, and Schatz[6] described them with a spheno-orbital meningioma. We report herein a clinicopathologic study of opticociliary shunt vessels in an adult with progressive visual loss, optic disk pallor, and proptosis caused by a primary optic nerve sheath meningioma of the orbit.

CASE REPORT

A 59-year-old man had a one-year history of blurred vision in his left eye. Visual loss increased gradually without pain. He had no other ocular or neurologic symptoms, and his past medical history and family history were unremarkable. On examination, visual acuity with best spectacle correction was 6/9 (20/30) in the right eye and no light perception in the left eye. The visual field of the right eye was normal by Goldmann perimetry.

Increased resistance to retrodisplacement of the left globe and 4 mm of proptosis were present. Extraocular movements were normal, except for slight limitation of abduction, supraduction, and infraduction of the left eye. An afferent pupillary defect was present on the left. Ophthalmoscopy of the right eye revealed no abnormalities. The left optic disk was flat and pale, with small tortuous, opticociliary shunt vessels near the margins at the 1, 2, 5, and 8:30 o'clock positions (Fig. 1). Fluorescein angiography indicated that these anomalous vessels filled during the retinal venous phase from branches of the central retinal vein and did not leak fluorescein dye (Fig. 2).

Routine x-rays of the skull and orbits revealed no abnormalities. Tomograms of the left optic canal indicated slight demineralization without enlargement. Computed axial tomography showed marked enlargement of the left optic nerve or its sheaths, or both, from the globe to the orbital apex, that enhanced moderately after intravenous injection of

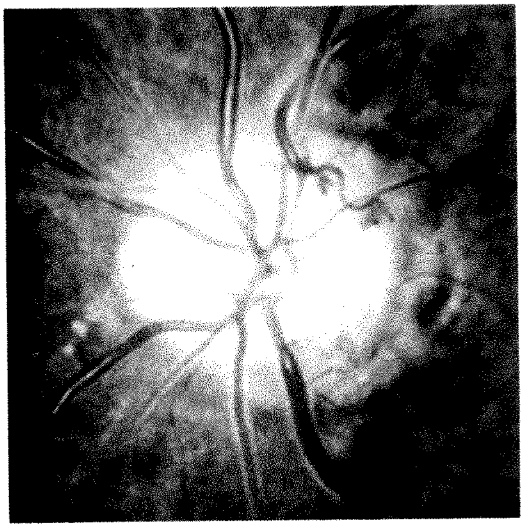

Fig. 1 (Zakka and associates). Fundus of the left optic disk showing pallor and dilated tortuous opticociliary shunt vessels at 1, 2, 5, and 8:30 o'clock positions.

From the Department of Ophthalmology (Drs. Zakka, Summerer, Yee) and the Department of Pathology (Dr. Foos), Jules Stein Eye Institute, UCLA School of Medicine; and the Department of Surgery, Division of Neurosurgery (Dr. Kim), Harbor General Hospital, Torrance. This study was presented before the Neuro-Ophthalmology Pathology, 10th Annual Symposium, Baltimore, Feb. 10, 1978. This study was supported in part by the Adelaide Stein Miller Research Fund and Public Health Research grants EY-00725 and EY-00331 from National Eye Institute, National Institutes of Health, Bethesda, Maryland.

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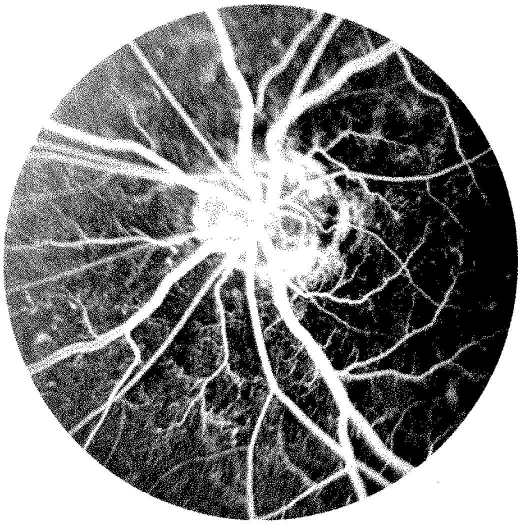

Fig. 2 (Zakka and associates). Fluorescein angiogram of the left eye during the retinal venous phase, showing filling of the opticociliary shunt vessels.

radiopaque dye (Fig. 3). No intracranial abnormalities were found. A diagnosis of primary optic nerve sheath meningioma without evident intracranial extension was made. Although we recommended observation of the orbital process until signs of definite intracranial extension developed, the patient requested complete excision of the neoplasm.

A left temporal craniotomy and removal of the roof of the left orbit were performed. Nodular thickening of the optic nerve sheaths extended from the back of the globe to the optic canal. The tumor was confined to the muscle cone, but it was closely applied to the back of the globe and could not be dissected from it. The left eye and tumor were excised en bloc. The intracranial portion of the left optic nerve was found to be atrophic, and the roof of the optic canal was thinned. The intracanalicular portion of the optic nerve was atrophic. The postoperative course was uneventful.

Gross examination showed an intact left eye, measuring 25.1 mm horizontally and 26.3 mm anteroposteriorly. The attached optic nerve measured 27.1 mm in length. It was encased by an irregular mass that ranged from 9 to 13 mm in diameter. A firm mushroom-shaped nodule protruded superiorly from the central aspect of the main tumor (Fig. 4). Horizontal sectioning and examination with the stereoscopic microscope revealed a markedly pale optic nerve head with abnormal vessels on the surface of the disk. These vessels were small, tortuous, and filled with blood; they represented the opticociliary shunt vessels that were observed clinically. The retinal nerve fiber layer was diffusely attenuated nasal and inferior to the optic disk, and the retinal veins were dilated.

Microscopic examination revealed almost total replacement of the optic nerve by a homogeneous, hypercellular mass within the dural sheaths. Occasional psammoma bodies and areas of focal calcification were found in the mass. Gliotic remnants of the optic nerve were compressed by the neoplasm (Fig. 5). The tumor cells were arranged in whorls and had poorly defined cell boundaries, finely granular cytoplasm, and oval vesicular nuclei (Fig. 6). No mitotic figures were found. These findings were indicative of a primary optic nerve sheath meningioma of the meningotheliomatous type.

The retrobulbar portion of the optic nerve immediately behind the globe showed marked widening and gliosis, but the tumor had not invaded the globe (Fig. 7). The central retinal vein and its branches

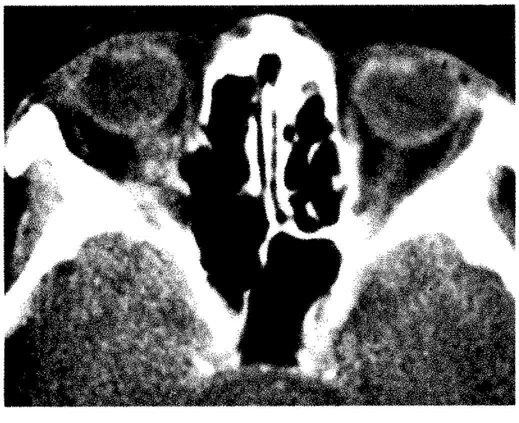

Fig. 3 (Zakka and associates). Computed axial tomogram showing marked enlargement of the left optic nerve, or its sheath, or both, from the globe to the orbital apex.

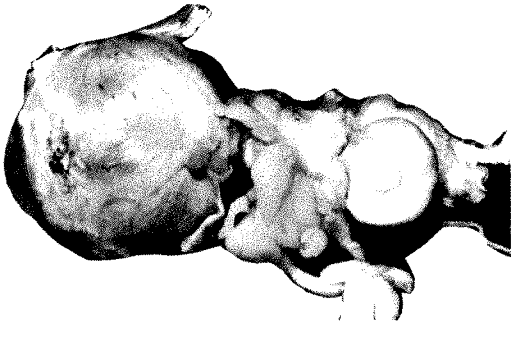

Fig. 4 (Zakka and associates). Left eye with attached optic nerve meningioma showing marked involvement of the nerve and adherence of the tumor to the posterior pole. Note the discrete mushroom-shaped nodule projecting superiorly from the optic nerve meningioma.

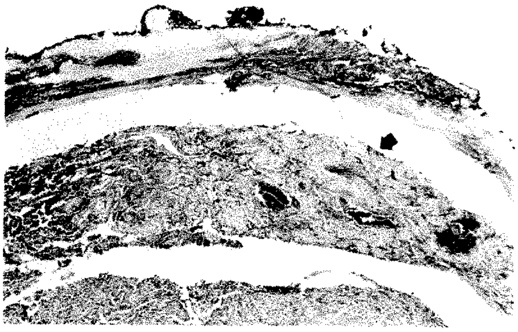

Fig. 5 (Zakka and associates). Cross-section of the left optic nerve 1 mm behind the globe. Arrow indicates gliotic remnants of the nerve that was compressed by the tumor (hematoxylin-eosin, ×50).

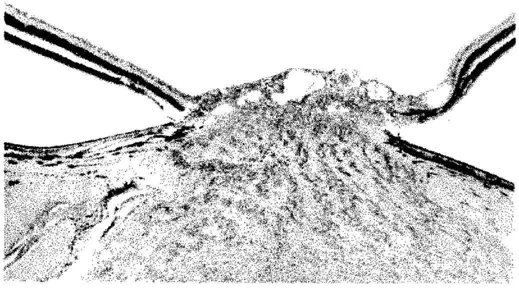

Fig. 7 (Zakka and associates). Horizontal section of the optic nerve and globe. There is marked gliosis and widening of the optic nerve immediately behind the globe. Several, dilated thin-walled vessels within the temporal margin of the disk extend toward the choroid (hematoxylin-eosin, ×50).

were dilated at the disk and in the adjacent retina, and the vein within the nerve was compressed. Step-level sectioning through the optic disk indicated that the small tortuous opticociliary vessels at the surface communicated with deeper thin-walled vessels within the disk (Fig. 8). A thin-walled vessel from the adjacent choroid was traced almost directly to these deeper channels, suggesting drainage of the anomalous surface vessels into the choroidal venous system (Fig. 9). The choroidal vessel was lined by endothelial cells, and it was filled with plasma and red blood cells.

Microscopic examination of the eye revealed atrophy of the nerve fiber layer and decreased numbers of ganglion cells. Several small microaneurysms of the retinal capillary network were also found, but no hemorrhages or exudates were present.

DISCUSSION

In 1898, Elschnig[5] described microscopic features of opticociliary shunt vessels in an adult with an optic nerve sheath meningioma of the orbit. He was able to trace these vessels to choroidal veins that drained into vortex veins. Rodrigues, Savino, and Schatz[6] described such vessels in association with a spheno-orbital meningioma and demonstrated opticociliary veins by fluorescein angiography and histopathologic examination. Similar

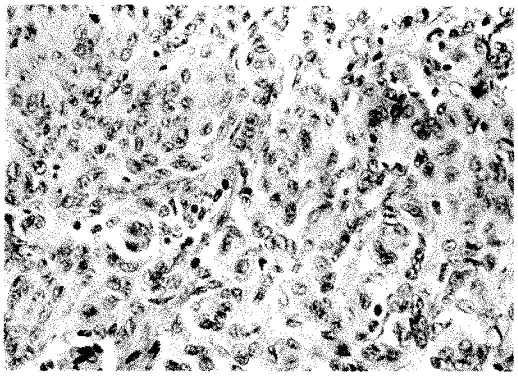

Fig. 6 (Zakka and associates). Cross-section of the optic nerve sheath tumor showing characteristic polygonal cells arranged in whorls with ill-defined cell borders, finely granular pale cytoplasm and oval, spherical nuclei containing conspicuous nucleoli (hematoxylin-eosin, ×500).

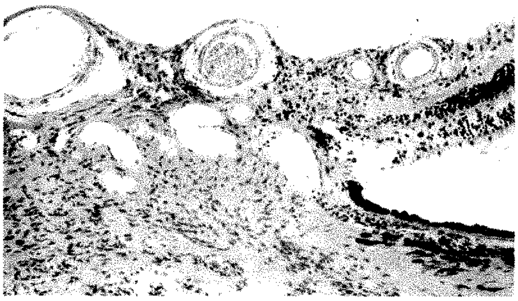

Fig. 8 (Zakka and associates). Horizontal section of the optic disk showing dilated branches of the central retinal vein within the retina and dilated thin-walled vessels within the nasal margin of the disk (hematoxylin-eosin, ×200).

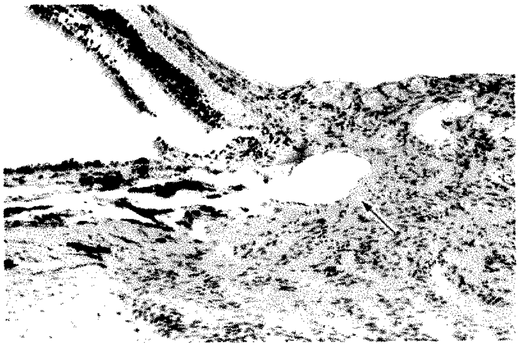

Fig. 9 (Zakka and associates). Horizontal section of the optic disk showing superficial dilated opticociliary shunt vessels near the temporal margin and a dilated endothelial-lined thin-walled vessel (arrow) extending from the disk into the choroid. There is an artifactual separation of tissue immediately posterior to this vessel (hematoxylin-eosin, ×200).

retinochoroidal shunt vessels have been described in many other ocular disorders.[7-12] Opticociliary shunt vessels are rarely present congenitally in association with other ocular defects, such as coloboma of the disk, or acquired in disorders causing obstruction of the central retinal vein, such as chronic papilledema, optic nerve glioma, perioptic vascular malformations, and central retinal vein occlusion. Frisen, Hoyt, and Tengroth,[3] Spencer,[13] and Hollenhorst, Hollenhorst, and MacCarty,[4] have emphasized that opticociliary shunt vessels associated with visual loss, proptosis, and optic disk pallor, are highly characteristic of primary and secondary optic nerve sheath meningiomas of the orbit. Presumably, intracranial meningiomas extending into the orbit along the optic nerve sheaths, and meningiomas arising from arachnoidal cells of the optic nerve sheaths compress the central retinal vein. Pre-existing vascular channels in the optic disk that interconnect the retinal venous and choroidal venous systems dilate to form the tortuous shunt vessels that are observed clinically.

The management of optic nerve sheath meningiomas originating primarily in the orbit is controversial.[14] In adults, the tumor grows slowly and has no significant malignant potential, but it can extend intracranially or into surrounding orbital tissues. In an adult with severe visual loss from an optic nerve sheath meningioma confined to the orbit, some investigators have advised observation without surgical intervention until radiologic or clinical evidence of intracranial extension is found or until severe exophthalmos develops.[2] Others advise surgical excision of the tumor in the orbit.[15,16] Optic nerve sheath meningiomas in children have a greater potential for rapid intracranial extension than do similar tumors in adults,[17] and early surgical treatment might be considered.

Hollenhorst, Hollenhorst, and Mac Carty[4] recently reemphasized and documented the poor visual prognosis when opticociliary shunt vessels were found associated with optic nerve sheath meningioma. In nine eyes with shunt vessels of eight patients who had adequate follow-up, all of the eyes had either severe permanent loss of central vision or no light perception. As indicated by the present case, when the tumor of the optic nerve sheath has enlarged sufficiently to obstruct the central retinal vein producing opticociliary shunt vessels, compression of the optic nerve is severe.

SUMMARY

A 59-year-old man developed painless visual loss in his left eye over a one-year period, with rapid progression to blindness over three months. Ocular examination of the left eye revealed proptosis, marked optic disk pallor, dilated retinal veins, and congeries of vessels at the disk margin (opticociliary veins). His right eye was unremarkable. Fluorescein angiography and histopathologic examination confirmed the presence of opticociliary veins communicating between branches of the central retinal vein and the adjacent choroidal vasculature. Primary orbital

optic nerve meningioma associated with opticociliary veins was diagnosed.

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NOTES, CASES AND INSTRUMENTS

HEMORRHAGE INTO RETINOSCHISIS DIAGNOSED AS MALIGNANT MELANOMA

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AND
JONATHAN FRATKIN, M.D.
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Hemorrhage into retinoschisis has rarely been noted clinically.[1] Presumably the hyalinized blood vessels with thickened walls typically found in areas of senile retinoschisis are resistant to rupture despite loss of surrounding retinal tissue. Several cases of blood-filled retinal macrocysts associated with retinal detachment and diagnosed as malignant melanoma have been reported.[2-4] We report herein a case of organized hemorrhage in retinoschisis that clinically and with ultrasonography was diagnosed as malignant melanoma.

CASE REPORT

A 55-year-old man had a malignant mixed tumor of the left parotid gland removed. He underwent radical neck surgery and a course of radiation to the left side. Eleven years later the patient was referred to us because of a superior temporal retinal lesion in his right amblyopic, exotropic eye. Visual acuity was R.E.: 6/60 (20/200) and L.E.: 6/9 (20/30). The diagnosis was bilateral retinoschisis. One year later the patient returned because of a mass lesion in the same superior temporal region of the right eye. Visual acuity was unchanged. The lesion was elevated into the vitreous, appeared dark, and did not transilluminate. A posterior subcapsular cataract prevented fundus photography and useful fluorescein angiography. Ultrasound examination revealed a tumor mass of medium reflectivity extending into the eye. The lesion was vascularized so that spontaneous spike movements were present. The diagnosis clinically and with ultrasonography was malignant melanoma.

The patient was scheduled for a subconjunctival radioactive phosphorus (^{32}P) uptake test, but he had a low platelet count of 53,000 cells/mm^3. Hematocrit and hemoglobin were normal. Surgery was therefore deferred but a transconjunctival ^{32}P uptake test was done, and the results were negative. A bone marrow biopsy revealed myeloid and megakaryocytic hyperplasia and the patient was transferred to hematology. No cause for the platelet deficiency was found. A splenectomy cured the platelet deficiency. A prostate biopsy performed at the time of the splenectomy revealed adenocarcinoma. A repeat ^{32}P uptake test was rejected by the nuclear medicine service as potentially hazardous. Repeat ultrasound examination again resulted in a diagnosis of malignant melanoma. The clinical diagnosis was unchanged and the right eye was enucleated two months after the original admission.

Gross examination of the globe showed that it did not transilluminate in the superior temporal quadrant. On sectioning, a solid fleshy tumor in the retina was visible. It was diagnosed as hemorrhage within the retina (Figs. 1 and 2).

Histologic examination showed that the tumor consisted of organized and unorganized hemorrhage into an area of retinoschisis (Fig. 3). Blood-bearing vessels were present in the area of organized hemorrhage (Fig. 3). The retinoschisis was most visible at the edge of the tumor (Figs. 1 and 2). Retina separated the tumor from the vitreous and pigment epithelium, showing the hemorrhage to be truly intraretinal.

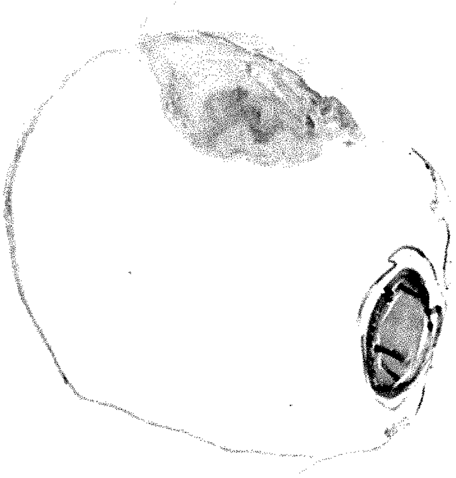

Fig. 1 (Cibis and Fratkin). Gross section of the right eye showing organized retinal hemorrhage into an area of retinoschisis superior temporally. The retinoschisis is most visible at the margins of the lesion.

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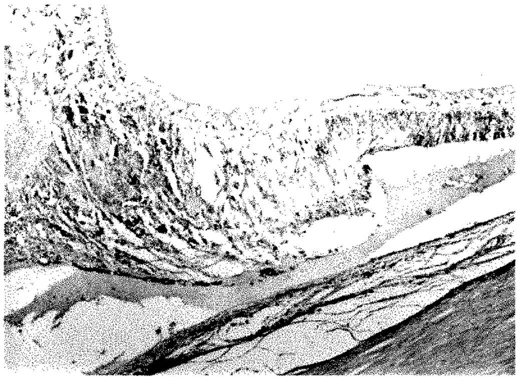

Fig. 2 (Cibis and Fratkin). Posterior edge of the hemorrhage into retinoschisis. The hemorrhage into schisis cavities is obvious.

There was a small subclinical retinal detachment beneath the tumor extending posteriorly.

DISCUSSION

To our knowledge, this is the first report of histopathology of hemorrhage in retinoschisis that clinically and with ultrasonography simulated a malignant melanoma. Misdiagnosis occurs when a lesion simulates the physical properties of a lesion, in this case malignant melanoma. The homogeneity of the hemorrhage gave the lesion reflectivity to ultrasound typical of melanoma. Its location and elevation into the globe and its vascularization with blood flow within the lesion producing spontaneous spike movements on the A-scan ultrasound were also typical of malignant melanoma and were misinterpreted. Clinically the color, elevation, and lack of light transmission gave the appearance of a melanoma. We believe that its homogeneous consistency and vascularity would have indicated malignant melanoma if fluorescein angiography had been done.

SUMMARY

A 55-year-old man had organized hemorrhage into retinoschisis that simulated malignant melanoma both clinically and with ultrasonography. The correct diagnosis was made histopathologically after enucleation.

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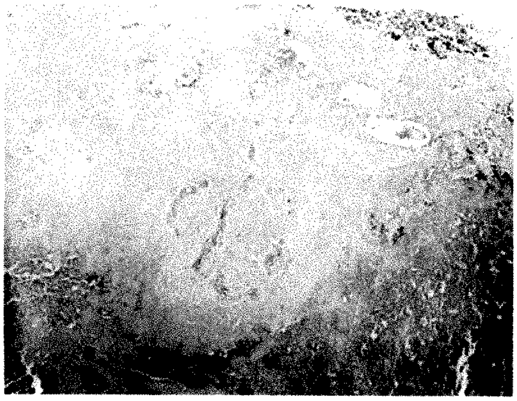

Fig. 3 (Cibis and Fratkin). Center of the tumor near the vitreous surface showing organized hemorrhage with blood-bearing vessels. The more obviously hemorrhagic portion inferiorly corresponds to the darker band running through the center of the lesion in Figure 1.

LEARNING SCLERAL DEPRESSION WITH BINOCULAR INDIRECT OPHTHALMOSCOPY

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Scleral depression with binocular indirect ophthalmoscopy is a difficult discipline to master. To obtain an image of the ocular fundus with the binocular indirect

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ophthalmoscope the examiner's pupil, the center of the hand-held condensing lens, and the patient's pupil must all be on a straight line. When scleral depression is performed, the tip of the scleral depressor must be the fourth point on the straight line. The patient's pupil becomes the center of rotation as the examiner's pupil and the lens move in one direction while the tip of the depressor is moved in the opposite direction.

The greatest difficulty is determining when the tip of the scleral depressor is on the straight line. The learning time for scleral depression can be significantly shortened by the use of a simple optical concept with which few ophthalmologists are familiar. The color of the pupillary reflex should be noted while looking through the ophthalmoscope without using the condensing lens, while the patient's eye is rotated away from the examiner. When the tip of the depressor is on the straight line, the color of the pupillary reflex will darken slightly (Figure). This phenomenon has been described[1] but never accurately illustrated. The actual scleral indentation cannot be seen through the pupil. It is only the optical effect of the indentation and darkening of the pupillary reflex that is visible when observed through the ophthalmoscope without the condensing lens. If the condensing lens is then moved into the proper position, the actual indentation from the depressor will be visible. This maneuver should be practiced without the condensing lens until the color of the pupillary reflex can be readily changed before attempting to visualize the scleral indentation through the condensing lens. This change in color of the pupillary light reflex can be observed in any eye, phakic or aphakic, regardless of the type or degree of ametropia.

The previous description is based on the assumption that the examiner has mastered the technique of binocular indirect ophthalmoscopy sufficiently in order to examine the equatorial zone of the retina. A lens of 18 to 20 diopters is preferred to one of 27 to 30 diopters because it can be held farther from the eye, which allows more room for the hand using the depressor. One of the most common errors for the beginner is depressing too anteriorly. Scleral depression in the area of the corneoscleral limbus can be dangerous. One need not be timid about pushing the tip of the scleral de-

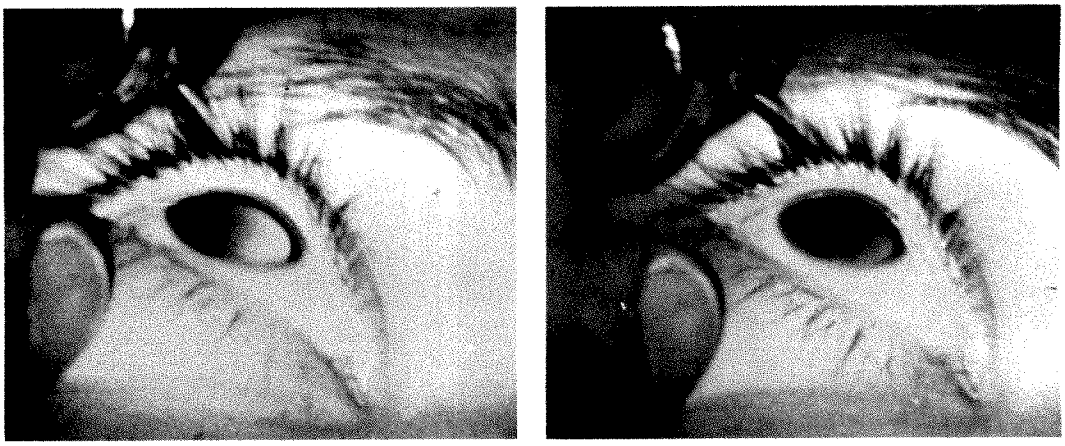

Figure (Kraushar). The pupillary reflex darkens when the tip of the depressor is on a straight line from the examiner's pupil through the patient's pupil. If the hand-held condensing lens is then positioned properly the scleral indentation will be visible.

pressor more deeply into the orbit to reach the area of the equator. Only light pressure is required for scleral depression and the amount of pressure will not change the color of the pupillary reflex if all four points are not on a straight line.

SUMMARY

Scleral depression with binocular indirect ophthalmoscopy will be easier to learn if the examiner observes the patient's pupillary reflex through the ophthalmoscope without looking through the hand-held condensing lens. When the tip of the scleral depressor is properly positioned the pupillary reflex will darken slightly. If the condensing lens is then moved into position the actual scleral depression will be visible.

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FIXATION OF CADAVER EYES FOR SURGICAL PRACTICE

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New ophthalmic skills and practice in placement of sutures and dissection of the eye may be obtained by use of cadaver eyes that are not suitable for transplantation or scleral preservation. I have found the following method to be most effective in providing material for this surgical practice.

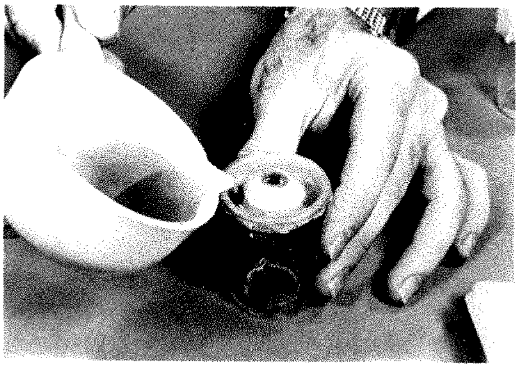

Figure (Silverman). Cadaver eye fixed with warmed dental wax in medicine cup.

A sheet of No. 7 dental wax is warmed under hot tap water and base-molded for receipt of the cadaver eye in a 1-ounce medicine glass. A No. 30 hypodermic syringe is passed into the cadaver eye through the optic nerve and saline is injected to restore the intraocular pressure to normal. A ligature around the optic nerve is tied as the needle is removed. The globe is then placed in the convexity formed in the medicine glass, and warmed liquid dental wax is poured around the globe where it rapidly sets, holding the eye in position (Figure).

To perform surgery, the medicine cup containing the eye is placed in a styrofoam wig stand. Openings for the cup are made by heating the medicine glass and allowing it to melt into the styrofoam. The back of the wig stand is cut off evenly to allow the head to lie flush.

SUMMARY

Cadaver eyes for surgical practice may be prepared by melting No. 7 dental wax in a 1-ounce medicine glass and fixing the globe with melted dental wax. The preparation is then placed in a styrofoam wig stand with openings prepared for the medicine glass.

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The opinions or assertions contained herein are the private views of the author and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

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MEETINGS, CONFERENCES, SYMPOSIA

EDITED BY THOMAS CHALKLEY, M.D.

AMERICAN ACADEMY OF OPHTHALMOLOGY 1978

Exciting and exhilarating are words that aptly describe the first independent meeting of the American Academy of Ophthalmology in the spacious Kansas City Convention Center Oct. 22-26, 1978. Under the statesmanlike leadership of Frederick C. Blodi, president of the American Academy of Ophthalmology and president-elect of the combined American Academy of Ophthalmology and Otolaryngology, and the extremely competent management of Bruce E. Spivey, executive vice president of the American Academy of Ophthalmology, the meeting was resoundingly successful.

The scientific program was most informative. It encompassed 419 scientific presentations, 573 instruction course hours, 39 scientific exhibits, 67 scientific poster presentations (a new feature of the annual meeting), and a total faculty of more than 1,000 ophthalmic scientists. The Academy secretaries, Robert D. Reinecke (Program), David Shoch and Melvin Rubin (Instruction), and David Paton (Continuing Education) are to be praised for planning this meeting.

For this return to the city where the organization was founded in 1896, the Academy attracted 9,608 registered participants. Moreover, the Academy assembly was greatly enriched by coordinated meetings of several dozen associate societies, national committees, and scientific organizations.

Eugene L. Derlacki, president of the combined American Academy of Ophthalmology and Otolaryngology, opened the meeting and presented the Academy Honor Award to 29 ophthalmologists. The Public Service Award was conferred on Mrs. Virginia Boyce for her valuable contributions to preservation of vision as Executive Secretary of the National Society to Prevent Blindness. A special Distinguished Service Award was presented with a standing ovation to C. M. Kos in recognition of his years of service to the Academy; and cordial comments of appreciation accompanied introduction of the Guests of Honor: Benjamin F. Boyd (Panama), David G. Cogan (United States), and Professor Jules François (Belgium).

Jules Stein presented the Research to Prevent Blindness Trustees Award for Outstanding Ophthalmic Achievement to Robert Machemer. This prestigious honor was awarded to Dr. Machemer for pioneering scientific work, which led to the introduction of pars plana victrectomy in 1970 and to subsequent development of this sight-restoring surgical procedure.

Professor Norman Ashton delivered the XXXV Edward Jackson Memorial Lecture, "Allergic Granulomatous Nodules of the Eyelid and Conjunctiva," published elsewhere in this issue of THE JOURNAL. An excellent symposium on the "Current Status of Retrolental Fibroplasia (RLF)" was dedicated to the late V. Everett Kinsey. Arnall Patz, symposium chairman, introduced reports on eye examination of the premature infant (Robert E. Kalina) histopathology of RLF (Norman Ashton), fluorescein angiography of RLF (John T. Flynn), complications of RLF (William S. Tasman), experimental observations on vitamin E in RLF (Dale Phelps), and clinical observations on vitamin E in RLF (David B. Schaffer). The symposium concluded by recommending, as minimum procedures, that all newborn infants with (1) history of supplemental oxygen, (2) gestational age of less than 36 weeks, or (3) birth weight of less than 1,900 g undergo an eye examination at the time of discharge from the hospital. If this examination demonstrates evidence of active RLF, frequent eye examinations at regular intervals thereafter are indicated to evaluate and manage the active disease.

Additional symposia throughout the meeting were concerned with ophthalmic drugs (Robert P. Burns, chairman), clinical management of physiologic myopia (Aran Safir, chairman), extraocular muscle problems associated with Graves' disease (Robert M. Day, chairman), congenital cataracts (Irene H. Maumenee, chairman), intraocular lenses and contact lenses (Jack Hartstein, chairman), neuro-ophthalmology (J. Lawton Smith, chairman), medicolegal problems in ophthalmology (Jerome W. Bettman, chairman), glaucoma (Steven M. Podos, chairman), strabismus (Joseph H. Calhoun, chairman), tumors of the eyelids and orbit (Bartley R. Frueh, chairman) contact lens and spectacle lens correction of aphakia (Oliver H. Dabezies, Jr., chairman), intraocular lenses (Robert C. Drews, chairman), and concepts of phacoemulsification (Charles D. Kelman, chairman).

This extensive and awesome array of scientific activities was lightened by the traditional President's Reception and the innovative clustering of all alumni groups under distinctive banners adjacent to the four-story garden of the modern Crown Center Hotel. The good fellowship created by convivial social gatherings was extended to the pre-dawn, mini-marathon "Run for Fun," which sent physician-joggers on a course past the major hotels throughout the city.

Contributing to the egalitarian spirit of free exchange and open communication, the joint Ophthalmology Recertification Committee conducted an open hearing on this complex and controversial subject. Following a summary description of the ophthalmology Performance Evaluated Education for Recertification program, the ophthalmology PEER program, an exceptionally lively hearing emphasized genuine concern about the need for recertification, representation of all elements of the profession on committees considering recertification, and potential problems associated with all forms of evaluation. Before any additional action on this important topic is taken, these and other issues will be fully evaluated by the Ophthalmology Recertification Committee and the widely representative Ophthalmology Recertification Advisory Committee, formed shortly before the Academy annual meeting. This hearing emphasized the ability of ophthalmologists to consider openly even the most difficult issues and to deal with these matters in the forum of full discussion.

For the future, a solid foundation was established at the annual Business Meeting. Members voted, in concert with colleagues in the American Academy of Otolaryngology, to dissolve the American Academy of Ophthalmology and Otolaryngology, Inc., and to establish the American Academy of Ophthalmology, Inc. Named to honorary fellowship were Professors J. Bock (Vienna), P. Bregeat (Paris), F. Hollwich (Muenster), and H. Sautter (Hamburg).

Officers for the American Academy of Ophthalmology, Inc., in 1979 are as follows: President, Frederick C. Blodi; President-Elect, Edward W. D. Norton; First Vice President, Kenneth C. Swan; Second Vice President, Goodwin M. Breinin; Third Vice President, Robert C. Drews; Executive Vice President, Bruce E. Spivey; Secretary for Program, Robert D. Reinecke; Secretary for Instruction, Melvin L. Rubin; Secretary for Continuing Education, David Paton; Editor, Stanley M. Truhlsen; Councillor (1979), Byron H. Demorest; Councillor (1980), Whitney G. Sampson; Councillor (1981), Marshall M. Parks; and Councillor (1982), Thomas D. Duane.

With guidance of these officers, plans are well underway for the next annual meeting of the Academy in San Francisco Nov. 5-9, 1979. In succeeding years, the Academy will move to different portions of the United States. The annual meeting is scheduled for New Orleans Nov. 13-17, 1980; Atlanta, Nov. 1-5, 1981; and San Francisco in 1982. The combined meeting of the American Academy of Ophthalmology and the XXIV International Congress of Ophthalmology in San Francisco, Oct. 31 - Nov. 5, 1982, is expected to be the largest scientific assembly, in terms of professional presentations and attendance, in the history of ophthalmology.

BRADLEY R. STRAATSMA

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NEW INITIATIVES IN THE WAR ON BLINDNESS

The year 1978 marked a turning point in the war on blindness. After years of discussion and planning the World Health Organization (WHO) launched its Program for the Prevention of Blindness, and the International Agency for the Prevention of Blindness (IAPB), WHO's nongovernmental helpmate, convened its first General Assembly.

These events came none too soon. Admittedly fragmentary data suggest 40 million people, 1% of the human race, are blind.[1] The cost, in social services, lost productivity, and human suffering is incalculable—especially since the greatest burden falls on developing countries, which can least afford it. Blindness rates in trachoma and onchocerciasis endemic areas are commonly four to 20 times that of the West. Millions of India's citizens require cataract surgery; hundreds of thousands of Asian children are blinded and die each year with xerophthalmia.

It is toward this excess of avoidable blindness that WHO and IAPB have properly directed their attention. The goal is to reduce national blindness rates to below 0.5%, and more ambitiously, rates of even the worst affected communities to below 1%.[2] Following the recommendation of an earlier Study Group,[3] blindness is being defined as vision less than 3/60 (unable to count fingers at 3 feet).[4]

By the very nature of the problem these goals are long term. The number of individuals already blind from xerophthalmia, trachoma, and onchocerciasis can decrease, and prevalence rates decline, only through aging and natural attrition. In the short run, effectiveness will be determined by the reduction in the numbers of new cases of disease and blindness.

WHO's new program is unique in providing a comprehensive, coordinated approach to the problem. The strategy is already clear: identify the major causes of blindness, their etiologic determinants and geographic distribution; provide intensive intervention to the worst affected communities; and develop integrated programs for dealing with the root causes, as well as established cases of these diseases.[4]

Assessment will usually entail regional or national prevalence surveys. Intensive intervention can take many forms: mobile surgical programs for cataract and entropion; distribution of massive dose vitamin A capsules for xerophthalmia, and the like. The thrust of the integrated programs is provision of eye services to traditionally neglected rural and urban communities. The primary health worker, often the only member of the health establishment to come in contact with a majority of the population, is ideally situated to recognize and treat early xerophthalmia and trachoma, supervise vitamin and antibiotic prophylaxis, encourage improved dietary and hygienic practices, and recognize and refer patients in need of more sophisticated medical treatment or surgery to nearby secondary facilities.

Tertiary facilities, traditional teaching hospitals with their complex, expensive equipment and western orientation have traditionally undercut these goals, by consuming a disproportionate share of available (and limited) funds and talent to provide highly sophisticated care to a tiny, privileged minority. Under the new scheme they will continue to provide high quality ophthalmic education, but together with training in nutrition, epidemiology, demography, and public health, so as to produce professionals better suited for developing and implementing new, community-oriented programs.

WHO's resources are limited. To a great degree initiative and financing must come from within each country. The association is playing a crucial role by encouraging formation of "national societies," 52 of which are now allied with it (44 sent delegates to the first General Assembly held in Oxford, England, last July); providing these societies with guidance and direction; and stimulating them to initiate programs, raise funds, and prod their governments into action.

Partially in response to these activities, wealthier countries are contributing to the emerging campaign: DANIDA, the Danish aid agency has provided $10 million to India's program: President Carter has designated blindness prevention a major priority of United States overseas assistance programs; and voluntary agencies, such as Helen Keller International, Royal Commonwealth Society for the Blind, and the International Eye Foundation have undertaken major, new initiatives in this area.

We can only hope this promising start will result in future better eye health around the world.

ALFRED SOMMER

REFERENCES

1. Prevention of Blindness. Report of the advisory meeting on programme development, Geneva, Feb. 20-23, 1978. WHO, PBL/78.4, 1978.
2. Strategic Planning for the Prevention of Blindness. Report of a Task Force, Woodstock, July 10-12, 1978. WHO, PBL/78.6, 1978.
3. The Prevention of Blindness. Report of a WHO Study Group. Tech. Rep. Ser. No. 518, Geneva, 1973.
4. Technical and Operational Approaches to the Prevention of Blindness. Report of a meeting. Asilomar, Oct. 8-13, 1978. In press.

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on $8^{1}/_{2}$ × 11-inch bond paper, with $1^{1}/_{2}$-inch margins on all four sides, and limited in length to two manuscript pages.

Instillation of Eyedrops

Editor:

The instillation of eyedrops is a difficult technique for many patients to mas-

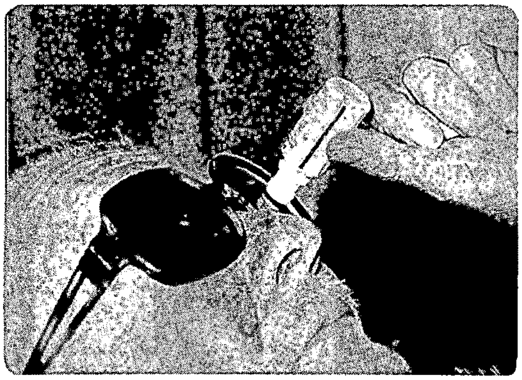

Figure (Tennant). Instillation of eyedrops through modified glasses.

ter. One of my patients has modified a pair of glasses to facilitate the maneuver. A hole is drilled in the lower half of each lens. The patient inserts the dropper into the hole, looks superiorly, and squeezes the bottle (Figure).

JEFFREY S. TENNANT, M.D.
Oak Lawn, Illinois

Helminthosporium Corneal Ulcers—Reply

Editor:

The letter to the editor of Michael R. McGinnis (Am. J. Ophthalmol. 86:853, 1978), a highly respected taxonomist, resulted in extensive reviews of the specimens from our cases and the published mycology reports.

In our paper we identified isolates as *Helminthosporium* species, by using the broad sense. This is a common practice among medical mycologists.[1] More extensive review of published material revealed that plant pathologists have subdivided this group. They now recognize the group *Drechsclera*, which has geniculate (bent-knee) conidiophores, as distinguished from the straight conidiophores of *Helminthosporium*. Remaining material was reexamined from our four cases from the viewpoint of separating those organisms with geniculate conidiophores into the *Drechsclera* group. The material from Cases 1 and 3 was not adequate to ascertain the genus of the fungus present. Cases 2 and 4 were identified as *Drechsclera.*

We appreciate Dr. McGinnis's insistance on precision, and recognize mycologic taxonomists as the final authorities in identifying fungal specimens. From a clinical standpoint, however, there are thus far no obvious divisions in manifestation or management between *Helminthosporium* and *Drechsclera.* Insistance on separating the two organisms might someday reveal clinical differences.

J. H. KRACHMER, M.D. AND
R. L. ANDERSON, M.D.
Iowa City, Iowa
P. S. BINDER, M.D.
San Diego, California
G. O. WARING, M.D.
Davis, California
J. J. ROWSEY, M.D.
Oklahoma City, Oklahoma
E. S. MEEKS, M.D.
Iowa City, Iowa

REFERENCE

1. Larone, D. H.: Medically Important Fungi. A Guide to Identification. Hagerstown, Harper and Row, 1976, p. 103.

BOOK REVIEWS

Field Guide to the Detection and Control of Xerophthalmia. By Alfred Sommer. Geneva, Switzerland, World Health Organization, 1978. Paperbound, 47 pages, 30 color figures. $5.80

Alfred Sommer, is an ophthalmologist trained in public health methods who is currently devoting his talents to preventing blindness from xerophthalmia in Indonesia. This brief guide is intended for clinicians, nurses, and public health officials involved in the diagnosis, treatment, and prevention of xerophthalmia, which accounts for 20,000 to 100,000 new cases of childhood blindness annually. The monograph describes the metabolism of vitamin A, and it contains a clinical application of xerophthalmia together with 30 colored figures of the disorder. A major discussion of the nature of the problem is followed by a method of preventing the disease. The guide concludes with appendices on examining the eyes and handling specimens in the field together with forms used in epidemiologic studies. The guide is well done and highly recommended reading for all.

FRANK W. NEWELL

Neuro-Ophthalmology Clinical Signs and Symptoms. By Thomas J. Walsh. Philadelphia, Lea Febiger, 1978. Hardcover, 294 pages, bibliography, table of contents, index, black and white figures; color figures. $22.50

Thomas Walsh, of Yale University, has written a neuro-ophthalmologic book expressly for residents and practitioners of ophthalmology. He believes they require a small manual on symptom diagnosis and management that tells how to do it without cluttering the mind with reference citations or pathophysiology. The text begins with papilledema and its differential diagnosis. This subject is handled in a brief chapter devoid of any illustrations of the critical ophthalmoscopic signs upon which diagnosis depends. Chapters discuss exopthalmos, blepharoptosis, the pupil, and diplopia, all without illustrations. In reading these chapters, the pragmatic expertise of the author is apparent, but the style and organization of his material seems more appropriate for a postgraduate course. Two chapters in the book, written by other contributors, are profusely illustrated and referenced. The text ends with more unillustrated chapters on blurred vision, headache, nystagmus, gaze, and field defects. This book is light reading and not without merit.

WILLIAM F. HOYT

OPHTHALMIC MINIATURE

It has always been a special type of patient that sought healing in cults: chronic patients as a rule and many of them neurotics. The symptoms of hysteria respond most easily to such treatment, but the cure is not a permanent one: the basic disease condition remains unaffected. It is very probable that Ambrosia left the temple seeing with both eyes, but it is quite possible that a few months or a few years later she became deaf or lame, or showed some other hysteric symptom.

Henry Sigerist
Civilization and Disease
Cornell University Press, 1943

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Acta Medica Scandinavica

HYPERTENSIVE EYE GROUND CHANGES: PREVALENCE, RELATION TO BLOOD PRESSURE AND PROGNOSTIC IMPORTANCE. Svärdsudd, K., Wedel, H., Aurell, E., and Tibblin, G. (Med. Dept., Sahlgren's Hosp., Gothenburg, Sweden). Acta Med. Scand. 204:159, 1978.

As part of a prospective population study of cardiovascular disease, ophthalmoscopic examination was performed in 855 randomly selected 50-year-old men in 1963. The examination was repeated four years later, in 1967. On both occasions hemorrhages, exudates and papillary edema were rarely seen. Attenuating arterioles, focal narrowing, crossing phenomena and broadened light reflex were all related to blood pressure but attenuating arterioles and/or focal narrowing discriminated blood pressure best. Isolated broadened reflex and/or crossing phenomena were not related to blood pressure. The change in blood pressure over four and ten years was the same in those who had and those who did not have hypertensive eye ground changes at the initial examination, indicating that eye ground changes do not precede hypertension. Mortality data and the morbidity in myocardial infarction and stroke were followed up until 1975. The importance of the separate eye ground variables for these mortality and morbidity end-points was analyzed, taking into account the importance of blood pressure, serum cholesterol and smoking. Focal narrowings were of importance for mortality, regardless of its cause, for stroke and for death from malignancy. Crossing phenomena were of importance for mortality, regardless of cause, for fatal coronary heart disease, for stroke and for deaths other than cardiovascular and cancer deaths. The eye ground variables are thus of differing importance for different end-points. Grouping them as in Keith and Wagener's or other classification systems, means a loss of information and should be avoided. (4 figures, 5 tables, 27 references)—Authors' abstract

Archives of Ophthalmology

CONSIDERATIONS RELATED TO "GOING METRIC". Sloan, L. L., and Enoch, J. (Baltimore, Md.). Arch. Ophthalmol. 96:1567, 1978.

There is great pressure for abandoning the English system of feet in recording visual acuity. It is customary to substitute the equivalent distance in meters so that 20/20 becomes in the metric system 6/6. The authors point out that as long as a change is going to be made it would be more appropriate to pick a different distance and they suggest four meters for distance acuity testing and 40 centimeters for near. They point out that 6 does not go into 20 easily while four does—making conversion much simpler, that four meters is a much easier distance to obtain in testing lanes than is six meters, and that a simple subtraction of a quarter diopter sphere from the refraction findings at four meters would make the retina conjugate with infinity. The advantages seem to be so obvious, it is a wonder that a movement is not already afoot to adopt the distances suggested.—David Shoch

SMALL MELANOMAS OF THE CHOROID. Barr, C. C., Sipperley, J. O., and Nicholson, D. H. (Bascom Palmer Eye Inst., Univ. Miami School Med., Miami, Fla.). Arch. Ophthalmol. 96:1580, 1978.

Clinical follow-up was possible on 36 patients who had melanomas of the choroid that were less than 10 mm in diameter and therefore qualified as "small melanomas." Of the 18 patients having a follow-up period of six years or more, two had tumor deaths yielding a death rate of 11%. The factor that correlated most clearly with tumor death was neither size nor Callender classification but the number of mitoses per high-powered field. Therefore the authors conclude that mitotic activity might be a reliable indicator of antecedent tumor growth. (1 figure, 2 tables, 13 references)—David Shoch

Bulletin of the New York Academy of Medicine

TREATMENT OF CHOROIDAL MELANOMAS. Abramson, D. H., and Ellsworth, R. M. (Manhattan Eye, Ear, Nose and Throat Hosp., New York, N.Y.). Bull. N.Y. Acad. Med. 54:849, 1978.

The development of A and B scans have now made melanomas identifiable even with opaque media. Further, in the last few years a great deal has been learned about the natural history of choroidal melanomas. With these advances a new regimen for the treatment of melanomas has been devised. If the melanoma is small, that is less than ten millimeters in diameter and less than two millimeters elevated, treatment may be either continued observation or photocoagulation or local radiation. This is particularly appropriate for a lesion in a patient's only eye.

Melanomas larger than ten millimeters in diameter and two millimeters in height may be classified as either medium or large. The authors classify those between ten and 15 mm in diameter as medium and those over 15 mm in diameter as large. However, the treatment is the same so this subclassification is not terribly important. The treatment for all these medium and large melanomas is enucleation unless the patient has only one eye in which case local radiation may be employed. The same is obviously true if the patient refuses enucleation. (25 references)—David Shoch

Geriatrics

HOW TO RECOGNIZE ISCHEMIC OPTIC NEUROPATHY - AND WHAT TO DO ABOUT IT. Glaser, J. S. (Dept. Ophthalmol., Bascom Palmer Eye Inst., Univ. Miami School Med., Miami, Fla.). Geriatrics 33:68, 1978.

Sudden or rapidly progressive monocular visual deficit associated with optic disk swelling signals onset of ischemic optic neuropathy. The peak incidence is usually in persons 60 to 70 years old. With the idiopathic (arteriosclerotic) type of this disease, pain and other nonvisual symptoms are atypical. Subsequent improvement in vision is rare, and afterwards the other eye becomes involved in about 40% of patients. It is vital not to subject these patients to invasive diagnostic procedures, particularly cerebral angiography. This syndrome should be recognized on the basis of clinical findings alone. Ischemic optic neuropathy due to cranial (temporal) arteritis occurs in an older age group (70 to 80) and is characterized by devastating visual loss, with weakness, weight loss, fever, chronic suboccipital headache, and pain or tenderness of the scalp of the forehead or temples. An elevated erythrocyte sedimentation rate is the most helpful laboratory finding in diagnosing arteritis. Positive results of temporal artery biopsy confirm the diagnosis, although a negative biopsy by no means rules it out. When cranial arteritis is not the suspected cause of ischemic optic neuropathy, therapy is controversial and results of corticosteroid treatment have been disappointing. (4 figures, 2 tables, 16 references)—Author's abstract

Journal of the American Medical Association

CONJUNCTIVITIS AND SKIN ERYTHEMA: OUTBREAK CAUSED BY A DAMAGED HIGH-INTENSITY LAMP. Halperin, W., Altman, R., Black, K., Marshall, F. J., and Goldfield, M. (Div. of Lab. and Epidemiology, New Jersey Dept. Health, Trenton, N.J.). J.A.M.A. 240: 1980, 1978.

Eighty-one members of girls' basketball teams were exposed to ultraviolet light while sitting in the bleachers of a school gymnasium. A mercury high-intensity discharge lamp lighting the bleacher area had a hole in its outer envelope that allowed the emission of ultraviolet light. Sixty-nine (85%) of the 81 girls were affected; 49 (71%) had symptoms of conjunctivitis and 63 (91%) had symptoms of erythema. (1 figure, 2 tables, 5 references)—Authors' abstract

Journal of Nervous and Mental Disease

PAPILLEDEMA FOLLOWING THERAPEUTIC DOSAGES OF LITHIUM CARBONATE. Lobo, A., Pilek, E., and Stokes, P. E.

(New York Hosp., Cornell Med. Ctr., New York, N.Y.). J. Nerv. Ment. Dis. 166:526, 1978.

A 29-year-old white woman was placed on lithium for psychiatric difficulties. Some nine months later she complained of some blurring of vision and examination revealed a bilateral papilledema. A complete physical examination including skull X-rays, brain scan and CT scan were negative and the patient was discharged from the hospital with a final diagnosis of "pseudotumor cerebri". At some point she was placed on dexamethasone as well but it was noted that the original diagnosis of papilledema was made when the only drug intake was lithium. This would appear to be another possible cause of the pseudotumor cerebri syndrome. (11 references)—David Shoch

Journal of Pediatric Ophthalmology and Strabismus

DIPHENYLHYDANTOIN TERATOGENICITY: OCULAR MANIFESTATIONS AND RELATED DEFORMITIES. Wilson, R. S., Smead, W., and Char, F. (Univ. Arkansas Med. Sciences, Little Rock, Ark.). J. Pediatr. Ophthalmol. Strab. 15:137, 1978.

A 12-year-old male child of an epileptic mother treated with primidone and diphenylhydantoin during pregnancy had multiple congenital ocular malformations including strabismus, blepharoptosis, hypertelorism, and wide epicanthal folds, all of which have been previously reported as manifestations of the recently described "hydantoin syndrome". He also exhibited trichomegaly, optic nerve abnormalities, and bilateral retinoschisis with maculopathy, which have not been previously reported in this syndrome. (5 figures, 1 table, 17 references)—Paul E. Romano

Laryngoscope

FACIAL PALSY: INTERPRETATION OF NEUROLOGIC FINDINGS. May, M., and Hardin, W. B., Jr. (Dept. Otolaryngol., Eye and Ear Hosp., Pittsburgh, Pa.). Laryngoscope 88:1352, 1978.

Five-hundred cases of facial paralysis are reviewed. Simultaneous bilateral facial palsy, associated lateral rectus weakness, slowly progressive facial weakness and facial weakness that showed no recovery after six months were considered to be incompatible with a diagnosis of Bell's palsy. In these cases an underlying cause was usually found, including tumor, trauma, vascular accident and specific inflammatory process. Recurrent ipsilateral palsy was usually indicative of Bell's palsy, but 30% of patients with that finding had tumors. Associated corneal hypesthesia preservation of forehead movement and altered facial sensation generally implied central lesions, but occurred in some cases of Bell's palsy. Seventy percent of patients with Bell's palsy had a complete spontaneous recovery. With associated decreased tearing, the prognosis for such recovery fell to 10%.

The authors propose a viral polyneuropathy as the cause of Bell's palsy, accounting for the variation in neurologic findings in this disease. (1 figure, 1 table, 17 references)—Gerald Harris

Mayo Clinic Proceedings

GUIDELINES FOR THE MANAGEMENT OF TRANSIENT ISCHEMIC ATTACKS. Sandok, B. A., Furlan, A. J., Whisnant, J. P., and Sundt, T. M. (Dept. Neurol., Mayo Clinic, Rochester, Minn.). Mayo Clin. Proc. 53:665, 1978.

On the basis of a review of the literature on anticoagulant, antiplatelet, and surgical treatment, supplemented by the authors experience, they have developed guidelines for the management of patients with transient ischemic attacks (TIA). 1. The majority of patients with vertebral-basilar TIA are treated medically. 2. If a skilled surgeon and an experienced angiographer are available, patients with typical carotid TIA who are suitable medical risks should have angiography followed by carotid endarterectomy if an appropriate lesion is found. 3. Nonoperated patients with TIA of less than two months' duration are treated with three months of warfarin therapy (unless contraindicated) before treatment with aspirin is begun. 4. Nonoperated patients with continuing TIA of two or more months' duration are treated with aspirin unless there has been a recent increase in the frequency, duration, or severity of TIA. Under these circumstances, warfarin therapy is advised for three months before aspirin is started. Aspirin therapy should be continued until the patient has been free of TIA for one year. 5. No treatment is advised for nonoperated patients whose last episode of TIA was longer than 12 months ago. (1 figure, 72 references)—Authors' abstract

Nature

TELEPHOTO LENS SYSTEM OF FALCONIFORM EYES. Snyder, A. W., and Miller, W. H. (Australian National Univ., Canberra, Australia). Nature 275:127, 1978.

Visual acuity in falconiform birds has been shown to be sharper than in man. Recently a falcon has been shown to have a grating detectability 2.6 times greater than that of man. However, the minimum intercone spacing of the falcon eye is only slightly less than the human retina. The explanation appears to lie in the presence of a spherical depression in the deep fovea of falconiform birds which acts as a negative lens component in a telescopic lens system. Therefore, the focal length of the bird's optical system can in theory exceed the axial length of the eye thus providing the relatively large images necessary for high resolving power in a localized region of the retina. (2 figures, 19 references)—David Shoch

Neurology

TOTAL BLINDNESS AFTER TRIVIAL FRONTAL HEAD TRAUMA: BILATERAL INDIRECT OPTIC NERVE INJURY. Venable, H. P., Wilson, S., Allan, W. C., and Prensky, A. L. (Dept. Ophthalmol., Homer Phillips Hosp., St. Louis, Mo.). Neurology 28:1066, 1978.

A ten-year-old girl developed bilateral blindness and partial third nerve paresis immediately following a closed head injury. Bilateral optic atrophy developed subsequently. This is the first report of an association between second and third nerve injuries after minor head trauma in the absence of a preexisting lesion. The pathophysiology of indirect injury to the optic nerve under these circumstances is uncertain, but the lesions in this patient seemed to be due to ischemia. (9 references)—Authors' abstract

Radiology

COMPUTED TOMOGRAPHY OF INTRAORBITAL OPTIC NERVE GLIOMAS IN CHILDREN. Byrd, S. E., Harwood-Nash, D. C., Fitz, C. R., Barry, J. F., and Rogovitz, D. M. (Hosp. for Sick Children, Div. Neuroradiology, Toronto, Ontario). Radiology 129:73, 1978.

Optic nerve gliomas were diagnosed with computed tomography (CT) in ten children. CT: (a) provides the best radiographic means of demonstrating the orbital portion of the optic nerves; (b) can evaluate the clinically unsuspected optic nerve for bilateral gliomas; (c) demonstrates gross intracranial involvement; and (d) can be used to follow the course of a glioma. Gliomas can present on CT as (a) a uniform thickening of the entire nerve; (b) a solitary fusiform enlargement of the nerve; or (c) an irregular solid thickening along the nerve. The authors routinely use axial CT of optic nerves as the primary diagnostic scan. (9 figures, 2 tables, 9 references)—Authors' abstract

OBLIQUE COMPUTED TOMOGRAPHY FOR ORBITAL MASS LESIONS. Komaki, S., Baba, H., and Matsuura, K. (Dept. Radiol., Kyushu Univ. Hosp., Maidashi, Fukuoka City, Japan). Radiology 129:79, 1978.

The CT scanners generally used for orbital lesions are designed to produce only transverse tomograms. These sometimes miss small lesions in the upper and lower portions of the orbit. To overcome this difficulty, the authors developed an oblique tomogram at a 45 degree angle using a standard neuroradiological CT scanner. Two cases are presented in one of which a mass was visible only on oblique tomogram and in the second a second tumor was revealed by the oblique scan. (3 figures, 3 references)—David Shoch

Science

EXTREME MYOPIA PRODUCED BY MODEST CHANGE IN EARLY VISUAL EXPERIENCE. Wallman, J., Turkel, J., and Trachtman, J. (Dept. Biol., City Univ. New York, New York, N.Y.). Science 201:1249, 1978.

It has been postulated that the accommodation and convergence associated with continuous near vision cause a change toward the myopic side in man. The authors tested this hypothesis in chicks by using two types of blinkers. In one kind the lateral field was obstructed so that vision was normal only straight ahead. It was assumed that this group would use a great deal of accommodation and convergence to see its food. The second group had one eye occluded so that it had only lateral vision. The unpatched eye was therefore used for feeding and other activities while the eye with the cylindrial occluder looked off in the distance. A third group of animals had one eye occluded. The first group whose vision was confined to the frontal visual field developed a myopia whose average was −10 diopters. The eyes whose vision was restricted to the lateral field had a mean refraction of −1.9 diopters. The third group—those with the translucent occluder—developed a myopia similar to the first group, an average of −12 diopters. Further, the eyes of the birds that were restricted to frontal vision had a significantly greater axial length than the other two groups. The authors suggest that the morphology of the eye is influenced not only by the absence of visual experience but also by the nature of the specific visual experiences. (3 figures, 17 references)—David Shoch

Transactions of the American Ophthalmological Society

VISUAL PROGNOSIS IN ADVANCED GLAUCOMA: A COMPARISON OF MEDICAL AND SURGICAL THERAPY FOR RETENTION OF VISION IN 101 EYES WITH ADVANCED GLAUCOMA. (Thesis) Kolker, A. E. (Washington Univ. School of Med., St. Louis, Mo.). Trans. Am. Ophthalmol. Soc. 75:539, 1977.

A detailed analysis of 101 eyes of 76 patients with advanced glaucomatous visual field loss but with retention of good visual acuity is presented. Patients were followed for a minimum of four years with an average duration of follow-up of 7.1 years. Loss of central vision, defined by permanent reduction of visual acuity to 6/60 (20/200), occurred with equal frequency in eyes treated medically (15.8%) or surgically (13.6%) for glaucoma. Sudden loss of central vision also occurred following cataract extraction (8.7%).

No patients lost central vision suddenly following surgery when central vision was spared at the time of operation. In addition, all cases that eventually lost central vision, either medically or surgically, demonstrated field defects which split fixation prior to its loss. Loss of central vision is seen rarely when medical therapy maintains the average intraocular pressure below 18 mm Hg, but increases markedly with higher pressures, reaching approximately 30% when average intraocular pressure is above 22 mm Hg. Progression in field loss is rare after successful glaucoma surgery, although cataracts develop in about 32% of such eyes. Cataracts also develop in 21% of medically treated eyes. In addition, about 50% of unoperated eyes demonstrate further field loss even when central vision is maintained.

In spite of very definite risks, serious consideration should be given to glaucoma filtering surgery when the intraocular pressure is consistently over 22 mm Hg in patients on medical therapy with advanced glaucoma. (5 figures, 9 tables, 10 references)—Author's abstract

MASSIVE PERIRETINAL PROLIFERATION: A LOGICAL APPROACH TO THERAPY. (Thesis) Machemer, R. (Bascom Palmer Eye Inst., Univ. of Miami School of Med., Miami, Fla.). Trans. Am. Ophthalmol. Soc. 75:556, 1977.

Massive periretinal proliferation, a serious complication of retinal surgery is caused by proliferation and fibrous metaplasia of cells mostly deriving from retinal pigment epithelium and retinal glial cells. Contracting fibrous membranes in the vitreous and on and also under the retina cause the intraocular changes of massive periretinal proliferation. Based on the improved understanding of this disease, a new therapeutic approach is presented, with the technique of pars plana vitreous surgery. Not only vitreous membranes but also preretinal membranes are removed. Forty-seven consecutive patients who suffered from severe massive periretinal proliferation were operated on with this technique and followed for six months or more. Seventeen patients (36%) had an attached retina. Twelve patients had a major visual improvement. The major postoperative complication is phthisis bulbi (10 eyes, 21%). (12 figures, 7 tables, 31 references)—Author's abstract

ADIE'S SYNDROME: SOME NEW OBSERVATIONS. (Thesis) Thompson, H. S. (Dept. Ophthalmol., Univ. Iowa, Iowa City, Ia.). Trans. Am. Ophthalmol. Soc. 75: 587, 1977.

The author has carefully studied 122 patients with Adie's syndrome. He reviews some of the classic findings in the literature and adds many new facts to the knowledge of this entity. For example, if the tonic pupils are bilateral, the chances are one in six that the patient has a positive serology; there is segmental paralysis and the segments are randomly distributed around the sphincter; most eyes with Adie's pupil have a slightly anesthetic cornea; and finally, there is a small but definite tendency for Adie's syndrome to become worse over the years; the second eye tends to become involved, the light reaction tends to lose further segments of the sphincter and the deep tendon reflexes may become slightly poorer. (20 figures, 8 tables, 41 references)—David Shoch

CLINICAL ASPECTS OF THE VISUALLY EVOKED POTENTIAL. (Thesis) Weinstein, G. W. (Univ. Texas Health Ctr., San Antonio, Tex.). Trans. Am. Ophthalmol. Soc. 75:627, 1977.

Generally speaking there is a close correlation between the visual evoked potential and the visual acuity. When the visual acuity is 6/9 (20/30) or better, the visual evoked potential is generally normal. There appear to be two chief uses for this test at this time. Patients with hysterical blindness manifest normal visual evoked potentials, while patients with amblyopia and organic causes of visual loss involving the higher visual pathways corresponding to the side of the lesion, show abnormalities of the visual evoked potential. The test is further useful in patients with opacities of the media in predicting whether vision might be improved with surgical intervention. The test that was most useful was the non-patterned flash. Patterned reversal stimulation is useful but this technique is easily supplanted by conventional visual acuity testing on almost every occasion when it might be employed clinically. (19 figures, 7 tables, 93 references)—David Shoch

Vision Research

SACCADIC OMISSION: WHY WE DO NOT SEE A GREY-OUT DURING A SACCADIC EYE MOVEMENT. Campbell, F. W., and Wurtz, R. H. (Physiological Lab., Univ. Cambridge, Cambridge, England). Vision Res. 18:1297, 1978.

Vision is interrupted several times a second by rapid eye movements (saccades) made from one point in the visual field to another. During these saccades the visual world sweeps across the retina and should produce a period of smeared vision lasting 25 to about 100 milliseconds depending on the size of the saccade, yet we normally do not see this smeared image of the visual screen generated by the eye movement. By using special lighting that was on only during eye movements, the authors were able to show that the lack of perception during saccadic eye movements, made in normal contoured environments, results primarily from the visual masking effect of a clear image before and/or after the eye movement acting on the grey-out during the eye movement. This "saccadic omission" is entirely a visual phenomenon and is far more powerful than the usually studied elevation of visual threshold for detection of a flash, "saccadic suppression". (2 figures, 30 references)—David Shoch

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

700 North Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least three months before the date of occurrence.

FRENCH OPHTHALMOLOGICAL SOCIETY: EIGHTY-FIFTH CONGRESS

The 85th Congress of the French Ophthalmological Society will be held in Paris May 6-10, 1979. The main theme will be on the vitreous. Other topics will include ocular immunology, trauma to the anterior segment of the eye, and paraclinical examinations in neuro-ophthalmology. For further information, write Professor P. Bec, C. H. U. Toulouse Rangueil, Chemin du Vallon, 31054 Toulouse, France.

INTERNATIONAL LENS IMPLANT COURSE

Professor C. D. Binkhorst will direct the International Lens Implant Course Sept. 5 and 6, 1979, at Elisabeth Hospital, Sluiskil, The Netherlands. For further information, write Ms. Mary van der Peijl, Abeelstraat 3, 4537, XR Terneuzen, The Netherlands.

MICHAIL M. KRASNOV: RECIPIENT OF LENIN PRIZE

Professor Michail M. Krasnov, a Soviet ophthalmologist, member of the International Academy of Ophthalmology, has been awarded a Lenin Prize for new developments in eye surgery including microsurgery and laser methods. The prize is the highest distinction for scientific achievements in the Soviet Union. It can be received only once in a person's life. It is the first time in history that the Lenin Prize was conferred on an eye specialist.

AMERICAN ACADEMY OF OPHTHALMOLOGY: NEW OFFICERS

New officers of the American Academy of Ophthalmology are as follows: Edward W. D. Norton, president-elect; Frederick C. Blodi, president; Kenneth C. Swan, first vice president; Goodwin M. Breinin, second vice president; Robert C. Drews, third vice president; Melvin L. Rubin, secretary for instruction; Thomas D. Duane, councillor. Bruce E. Spivey continues as executive vice president, and Stanley M. Truhlsen continues as editor of the Academy's journal, "Ophthalmology." Continuing on the board of directors are: Frank W. Newell, Kenneth L. Roper, and Bradley R. Straatsma. Reelected councillors are: Byron H. Demorest, Marshall M. Parks, and Whitney G. Sampson.

TENTH ANNUAL WILLIAM L. BENEDICT LECTURE

The Tenth Annual William L. Benedict Lecture, sponsored by The American Journal of Ophthalmology, was presented to the Minnesota Academy of Ophthalmology and Oto-Laryngology Jan. 12, 1979. Sadek K. Hilal of Columbia University, New York, spoke on "Computerized Tomography of the Orbit and Sinuses." Dr. Benedict was president of the Ophthalmic Publishing Company at the time of his death in 1969.

UNIVERSITY OF CHICAGO: ANNUAL ALUMNI DAY

The Department of Ophthalmology of the University of Chicago holds its Annual Alumni Day March 7, 1979. The guest speaker is Albert M. Potts, chairman, Department of Ophthalmology, University of Louisville. The staff and residents of departments of ophthalmology, alumni, and other physicians are invited. The full-day session will include a scientific program, a buffet luncheon, and reception. Registration and scientific session are scheduled to begin at 9 AM in P-117, Albert Merritt Billings Hospital, Chicago, Illinois. There is no charge. For further information, write Mrs. Karin Cassel, Department of Ophthalmology, 950 E. 59th St., Chicago, IL 60637; telephone (312) 947-6063.

MEDICAL COLLEGE OF GEORGIA: SELECTIVE TOPICS IN OPHTHALMOLOGY PROGRAM

The Department of Ophthalmology, Medical College of Georgia, will sponsor a three-day continuing education program, "Selected Topics in Ophthalmology," at the Savannah Inn and Country Club in Savannah, Georgia, March 26-28, 1979. Guest speakers include Frederick C. Blodi, Bruce Spivey, and Bradley Straatsma. Topics to be discussed include: orbital and ocular tumors, retinal disease, intraocular lenses, orbital problems following enucleation, and genetics and strabismus. A round table discussion, "Current and Future Status of Ophthalmology in the United States," will be held each day with the guest speakers as featured panelists. For further information, write the Division of Continuing Education, Medical College of Georgia, Augusta, GA 30901; telephone (404) 828-3967.

UNIVERSITY OF NEW MEXICO: FIFTH ANNUAL ROCKY MOUNTAIN NEURO-OPHTHALMOLOGY CONFERENCE

The University of New Mexico, Department of Neurology, will sponsor the Fifth Annual Rocky Mountain Neuro-Ophthalmology Conference in Jackson Hole/Teton Village, Wyoming, Feb. 21-23, 1979. For further information, write James L. Duran, Coordinator, Office of Continuing Medical Education, University of New Mexico, School of Medicine, Albuquerque, NM 87131.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY: ANNUAL MEETING

The Pittsburgh Ophthalmological Society will hold its annual meeting March 30 and 31, 1979, at the Pittsburgh Hilton Hotel. The central theme will be, "Practical Ophthalmology." The fee is $100 for members; $125 for nonmembers; and $50 for residents, which includes two breakfasts, lunch, and a banquet dinner March 30. For further information, write Franklin Cignetti, M.D., 2545 Mosside Blvd., Pittsburgh, PA 15146.

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO: POSTGRADUATE PROGRAM

The University of California, San Francisco, will sponsor a postgraduate program, "Human Chlamydial Infections and Associated Sexually Transmitted Diseases," Feb. 21-23, 1979, at Cole Hall, Medical Sciences Building, San Francisco, California. The fee, which includes box lunch, is $175; $100 for residents, interns, and postdoctoral students with a letter from their chief. For applications and further information, write Extended Programs in Medical Education, University of California, 1308 3rd Avenue, San Francisco, CA 94143.

OREGON ACADEMY OF OPHTHALMOLOGY: POSTGRADUATE CONVENTION

The Oregon Academy of Ophthalmology will hold its 38th Annual Postgraduate Convention March 16 and 17, 1979, at the Sheraton-Portland in Lloyd Center, Portland, Oregon. The fee for academy members is $115 before March 9, $125 thereafter; $140 for nonmembers before March 9, $150 thereafter. The fee includes lunchs and the banquet. Residents will be admitted without charge upon presentation of a letter from their department head. For further information, write Miss Rebecca Tarshis, Executive Secretary, 918 N.E. 44th Ave., Portland, OR 97213.

WILLS EYE HOSPITAL: FIFTH ANNUAL CONTACT LENS CONFERENCE

The Fifth Annual Contact Lens Conference will be held June 7-9, 1979, at the Caesar's World Boardwalk Regency Hotel, Atlantic City, New Jersey. The fee for the course is $175 for practitioners; no charge for residents with a letter from their department head. For further information, write Kenneth I. Michaile, M.D., 1930 Chestnut St., Philadelphia, PA 19103.

THE SOCIETY OF AIR FORCE CLINICAL SURGEONS: SEMINAR

The Society of Air Force Clinical Surgeons will hold a surgical postgraduate seminar April 22-25, 1979, at the Convention Center, San Antonio, Texas. Civilian physicians are encouraged to attend. For further information, write Robert Gilmore, M.D., Wilford Hall USAF Medical Center/SGHSG, Lackland AFB, TX 78236.

PERSONALS

J. TERRY ERNEST

J. Terry Ernest received the Heed Award during the 1978 American Academy of Ophthalmology meeting in Kansas City. The award was presented by Frank W. Newell, chairman of the Board of Directors of the Heed Foundation. Dr. Ernest received the award in recognition of his leadership and contributions in the field of ophthalmology. Dr. Ernest is professor of ophthalmology at the University of Wisconsin, where he was awarded the Research to Prevent Blindness Eye Research Professorship. Former recipients of the Heed Award include: Stuart I. Brown, Matthew D. Davis, Milton Flocks, Froncie A. Gutman, Eugene M. Helveston, William T. Hoyt, Philip Knapp, Paul R. Lichter, Harvey A. Lincoff, William Spencer, and William Tasman.

H. DUNBAR HOSKINS

H. Dunbar Hoskins of San Francisco was elected secretary-treasurer of the Pan-American Association of Ophthalmology. He succeeds Robert C. Drews.

ALAN LATIES

Alan Laties, professor of ophthalmology at the University of Pennsylvania, received the annual Humanitarian Award from the National Retinitis Pigmentosa Foundation in recognition of devoted services in the development and implementation of the research activities for this health agency. As chairman of the Retinitis Pigmentosa Scientific Advisory Board, Dr. Laties spearheaded program of research and clinical activities sponsored by the foundation.

JAMES B. MILLER

James B. Miller gave the Second James E. McDonald Memorial Lecture at Loyola University Stritch School of Medicine, Oct. 27, 1978. The title of his lecture was, "Carbon Dioxide Laser Photocoagulation. Photocautery during Vitreous Surgery."

FIGHT FOR SIGHT, INC., AWARDS

The chairman of the Scientific Advisory Committee, Fight for Sight, Inc., Charles A. Perea, announced that the support extended for the current year includes: 24 grants-in-aid; 13 postdoctoral research fellowships; 11 student fellowships; three departmental awards; four clinical research service grants; eight travel awards, with the possibility of two postdoctoral research fellowships and two grants-in-aid, pending support from the National Institutes of Health.

The closing date for applications for the 1979-1980 awards is March 1, 1979.

Members of the Scientific Advisory Committee are as follows: Bernard Becker, Goodwin M. Breinin, Charles J. Campbell, Claes Dohlman, Thomas D. Duane, Morton F. Goldberg, Paul Henkind, Herbert E. Kaufman, Arthur Keeney, Carl Kupfer, Alan M. Laties, Irving H. Leopold, A. Edward Maumenee, Frank W. Newell, Edward W. D. Norton, G. Richard O'Connor, Steven M. Podos, Harris Ripps, Samuel L. Saltzman, Harold G. Scheie, and Bradley R. Straatsma.

FIGHT FOR SIGHT, INC., AWARDS
1978-1979

GRANTS-IN-AID

INVESTIGATOR AND INSTITUTION	PROJECT TITLE	AMOUNT AWARDED
Gary W. Abrams, M.D. Bascom Palmer Eye Institute University of Miami Miami, Florida	Ophthalmic ultrasound laboratory	$15,000
Daniel Branton, Ph.D. Harvard University Cambridge, Massachusetts	Isolation of plasma membrane from bufo rod outer segments	4,852
Janice Burke, Ph.D. University of Arizona Health Sciences Center Tucson, Arizona	Vitreal cell cultures. Growth and macromolecule synthesis	6,400
William H. Dobelle, Ph.D. Columbia University New York, New York	Subject testing booth. Artificial vision experiments	7,500
Curtis D. Eckhert, Ph.D. Stanford University Stanford, California	Role of zinc in ocular development and function	7,735
Jay M. Enoch, Ph.D. University of Florida Gainesville, Florida	The evaluation of binocular vision in infants with normal and abnormal vision	4,410
Shao-Ling Fong, Ph.D. Baylor College of Medicine Houston, Texas	Enzymes of the visual cycle	3,340
Roland A. Giolli, Ph.D. University of California Irvine, California	Plasticity of axonal collaterals and terminals in the mammalian visual system	6,323
Sheldon R. Gordon*, B.S. Wayne State University Detroit, MI	Macromolecular changes in regenerating corneal endothelium	4,585
Ronald Klein, M.D. University of Wisconsin Madison, Wisconsin	Blood-ocular barriers under stress in experimental diabetes	5,068

*Continuation Award.

INVESTIGATOR AND INSTITUTION	PROJECT TITLE	AMOUNT AWARDED
James C. Liang*, M.D. University of Illinois Chicago, Illinois	Immunologic aspects of diabetic retinopathy	$6,500
Carlos Lopez, Ph.D. Sloan-Kettering Cancer Research Institute New York, New York	Immunology of herpes keratitis	5,500
Travis A. Meredith, M.D. Medical College of Wisconsin Milwaukee, Wisconsin	Mixed lymphocyte culture histocompatibility antigen typing in presumed ocular histoplasmosis	6,000
Charles W. Nichols, M.D. Scheie Eye Institute Philadelphia, Pennsylvania	Tracer dye studies of the blood retinal barrier in experimental diabetes	4,880
Jerry Y. Niederkorn, Ph.D. University of Texas - Southwestern Medical School Dallas, Texas	Humoral antibody-mediated and macrophage-mediated immunity in a feline model of ocular melanoma	6,934
Jang O. Oh, M.D. University of California San Francisco, California	Immunopathologic study of recurrent herpes simplex	5,000
Randall Olson, M.D. Louisiana State University New Orleans, Louisiana	Intraocular lens coatings and endothelial damage	5,000
Robert Ritch, M.D. Mount Sinai School of Medicine New York, New York	Localization of adenyl cyclase in the anterior chamber	4,850
Jerry W. Smith, Ph.D. Louisiana State University New Orleans, Louisiana	Viral antigens associated with herpetic eye disease	4,550
Richard A. Stone, M.D. Scheie Eye Institute Philadelphia, Pennsylvania	The measurement of redox states in the optic disk	6,260
Hugh R. Taylor, M.D. Johns Hopkins University Baltimore, MD	An animal model for trachoma	5,100
George O. Waring, M.D. University of California Davis, California	Comparison of five methods of measuring human corneal endothelial cells phographed by specular microscopy	8,558
Eugene J. Wenk,* Ph.D. New York Medical College Valhalla, New York	Cation localization within the lens using X-ray microprobe analysis	5,000
Michael E. Yablonski, M.D. Mount Sinai School of Medicine New York, New York	The measurement of aqueous flow in humans	5,066

*Continuation Award.

POSTDOCTORAL RESEARCH FELLOWSHIPS

INVESTIGATOR AND INSTITUTION	DIRECT SUPERVISOR	PROJECT TITLE	AMOUNT AWARDED
Fabian Abraham, M.D. University of Michigan Ann Arbor, Michigan	Dr. Mathew Alpern	Regional differences in the physiology of human retinal adaptation in health and disease	$8,000
Dan Agness, M.D. University of Miami Miami, Florida	Dr. Douglas Anderson	Therapy for neovascular glaucoma	8,000
Jonathan Dutton, M.D. Washington University St. Louis, Missouri	Dr. Theodore Krupin	Ocular transport of the cyclic nucletides	7,500
Robert Fenzl, M.D. University of Iowa Iowa City, Iowa	Dr. Jay Krachmer	The genetic transmission of keratoconus	8,000
Geoffrey Gold, Ph.D. University of California San Francisco, California	Dr. Juan Korenbrot	Detection of calcium release from rhodopsin-phospholipid vesicles	2,600 (Supplemental support)
Ivan Goldberg, M.D. Washington University St. Louis, Missouri	Dr. Michael Kass	Systemic factors influencing the development of glaucomatous optic nerve damage	8,000
Harry Kolodner, M.D. Louisiana State University New Orleans, Louisiana	Dr. Randall Olson	Prevention of endothelial cell damage with PVP (polyvinylpyrrolidone) coated intraocular lenses	3,335 (Short term)
Robert Pagon, M.D. University of Washington Seattle, Washington	Dr. Robert Kalina	Ophthalmologic genetics	6,500
Ajay Pande, Ph.D. Columbia University New York, New York	Dr. Abraham Spector	The covalent linkage of lipid and sugar to human structural lens protein and its relationship to senile cataract formation	8,000
Jaime Roizenblatt, M.D. University of California Los Angeles, California	Dr. Bradley Straatsma	Heredofamilial study of degenerative retinoschisis	7,000
Michael Rudy, M.D. University of Rochester Rochester, New York	Dr. Seymour Zigman	Enhancement of ocular tissue damage in aphakia by radiant energy	5,000

INVESTIGATOR AND INSTITUTION	DIRECT SUPERVISOR	PROJECT TITLE	AMOUNT AWARDED
Paul Singer, M.D. Washington University St. Louis, Missouri	Dr. Stephen Waltman	Vitreous flurophotometry in diabetes mellitus	$8,000
Robert Schumer, Ph.D. Bell Laboratories Murray Hill, New Jersey	Dr. Bela Julesz	Psychophysical investigation of the neural basis for binocular vision	4,000

STUDENT FELLOWSHIPS

INVESTIGATOR AND INSTITUTION	DIRECT SUPERVISOR	PROJECT TITLE	AMOUNT AWARDED
Barbara J. Baldwin University of Pennsylvania Philadelphia, Pennsylvania (Burroughs Wellcome Co. Fund	Dr. Herbert A. Blough	Biochemical studies on the fusion factor of herpesvirus	$900
Sherry Bressler Mount Sinai School of Medicine New York, New York (Burroughs Wellcome Co. Fund)	Dr. Robert Ritch	The role of foreign body giant cells in giant cell arteritis	900
Alan J. Deese University of California Santa Cruz, California (James Thurber Memorial Fund)	Dr. Edward Dratz	Lipid protein interactions and lipid dynamics in native retinal rod membranes	900
Paula C. Dobbs Massachusetts Eye and Ear Infirmary Boston, Massachusetts (Abraham Silver Memorial Fund)	Dr. David L. Epstein	A study of human ciliary body carbonid anhydrase in relationship to glaucoma	800
Marc H. Effron* Harvard Medical School Massachusetts Eye and Ear Infirmary Boston, Massachusetts (Burroughs Wellcome Co. Fund)	Dr. Eliot L. Berson	Small-field, receptor-isolated ERGs in hereditary retinal disease	900
Andrew T. Ishida University of California Los Angeles, California	Dr. Gordon L. Fain	Biochemical characterization of photoreceptor-bipolar-ganglion cell pathways in vertebrate retina	900
Jerome R. Kuszak Wayne State University Detroit, Michigan	Dr. Harry Maisel	The structure of the fiber cells membrane of the chick lens	900
Roger W. Li Boston University School of Medicine Boston, Massachusetts	Dr. Allan Kupferman	Topical Antibiotic Therapy of Staphylococcal Keratitis	700

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*Continuation Award.

INVESTIGATOR AND INSTITUTION	DIRECT SUPERVISOR	PROJECT TITLE	AMOUNT AWARDED
Karen L. Schneider Manhattan Eye, Ear and Throat Hospital New York, New York (Bob Hope Fund)	Dr. Lawrence Yannuzzi	A complete retrospective study of retinal branch vein occlusions treated by photocoagulation	$600
Paul Sternberg Jr. University of Chicago Chicago, Illinois	Dr. Ramesh C. Tripathi	Study of the pathophysiology of experimental hyphema	900
Rosalyn Weller Vanderbilt University Nashville, Tennessee (Burroughs Wellcome Co. Fund)	Dr. Jon H. Kaas	Selective loss of retinal ganglion cells after lesions of visual cortex	600

DEPARTMENTAL AWARDS

INSTITUTION	CHIEF OF DEPARTMENT	AMOUNT AWARDED
Texas Tech University School of Medicine Lubbock, Texas	James Price, M.D.	$7,500
University of North Carolina Chapel Hill, North Carolina	David E. Eifrig, M.D.	5,000
University of Texas San Antonio, Texas	George W. Weinstein, M.D.	4,800

CLINICAL RESEARCH SERVICE PROJECTS

Continuation Awards to the Fight For Sight Children's Eye Clinics to help defray the annual maintenance of these facilities.

Columbia-Presbyterian Medical Center, New York City—Philip Knapp, M.D., Director (Established 1961)	$16,376
Eye Institute of New Jersey, New Jersey College of Medicine, Newark—Anthony R. Caputo, M.D., Director (Established 1970)	22,130
Eye and Ear Hospital of Pittsburgh—David A. Hiles, M.D., Director (Established 1967)	14,207
The Wills Eye Hospital, Philadelphia—Donelson R. Manley, M.D., and Joseph H. Calhoun, M.D., Co-Directors (Established 1964)	35,625

TRAVEL AWARDS

This facet of the Fight For Sight research program was initiated in 1975 through a grant to the organization by the House of Vision, Inc., Chicago. These awards are to defray travel and per diem costs (in whole or in part), and are intended primarily to assist residents in ophthalmology with a minimum of one year's training who are authors or co-authors of a paper to be presented at a scientific meeting, or to participate in a symposium or seminar to which the candidate may make a significant contribution.

INVESTIGATOR	INSTITUTION	AMOUNT AWARDED
Jay Fleischman, M.D.	Johns Hopkins University Baltimore, Maryland	$400

INVESTIGATOR	INSTITUTION	AMOUNT AWARDED
Barrett G. Haik, M.D.	Columbia University New York, New York	$463
Lawrence W. Hirst, M.D.	Johns Hopkins University Baltimore, Maryland	400
Walter Jay, M.D.	University of Chicago Chicago, Illinois	493
Kenneth B. Mitchell, M.D.	Washington University St. Louis, Missouri	430
Charles W. Mohler, M.D.	Johns Hopkins University Baltimore, Maryland	400
Neal A. Sher, M.D.	University of Minnesota Minneapolis, Minnesota	500
Robert C. Snip, M.D.	Johns Hopkins University Baltimore, Maryland	400

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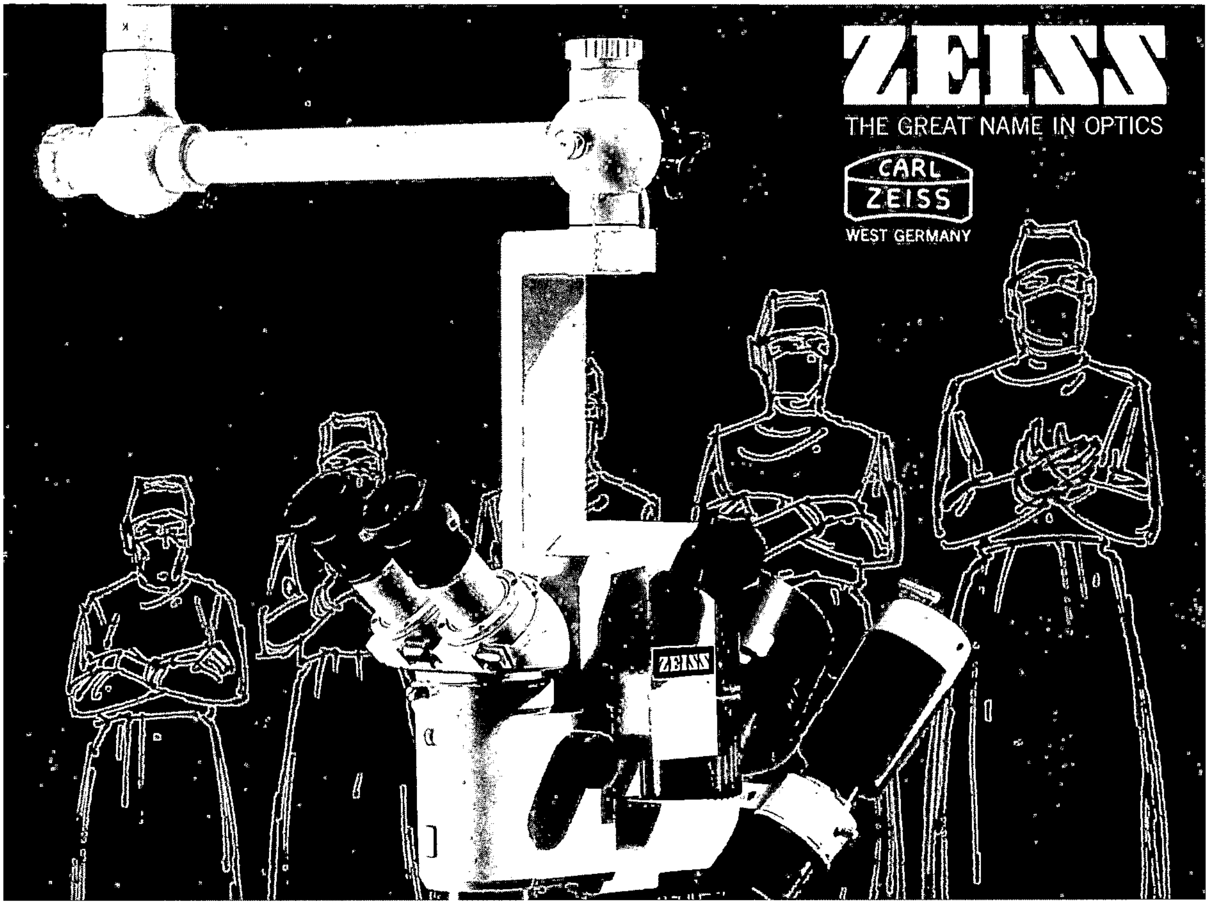

AMERICAN INTRA-OCULAR IMPLANT SOCIETY
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Kenneth J. Hoffer, M.D., Chairman

April 8-11, 1979 Century Plaza Hotel, Los Angeles, California

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Robert Azar
Kenneth Barasch
Ronald Barnet
C. D. Binkhorst
Elliott Blaydes
Herve Byron
William Cain
Lawrence Castleman
D. Peter Choyce
Henry Clayman
Gilbert Cleasby
John Corboy
Charles Cozean
Robert Drews
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James Salz
Ronald Schachar
H. John Shammas
Steven Shearing
John Sheets
Dennis Shepard
David Sholitan
William Simcoe
Norman Stahl
Daniel Taylor
Jerald Tennant
Barrie Thrasher
Walter Vukcevich
Doug Williamson
Robert Wolters
Scott Wong
Jan Worst

PAPERS TO BE PRESENTED partial listing

Mark VIII IOL vs. Mark IX IOL: 100 Case Comparison
Binkhorst IOL's vs. Choyce IOLs vs. Shearing IOLs
Primary vs. Secondary Insertion of Anterior Chamber IOLs: A Prospective Study
Endothelial Cell Count in Eyes with Anterior Chamber Lenses: 5 Year Study
Terry Keratometer in Intraocular Surgery
Slit Lamp Anterior Chamber Fluorophotometry in IOL Patients
Capsular Fixation with Secondary Implants
Loop Position: Hearing IOL
PETP Suture for Fixation of IOL Implants
New Approaches to Management of the Posterior Capsule with IOL Implantation
Endothelial Cell Count Studies After PKE and Insertion of a Kelman Lens
Prevention/Treatment of Posterior Capsule Opacification
Removal of IOL Using Two Lens Glides
New Techniques for Secondary Implantation
Contraindications to Lens Implant in Traumatic Cataracts: 144 Cases
Pediatric Endothelial Cell Studies
Iris Supported IOLs in Eyes with Primary Glaucoma: 40 Cases
Goniscopic, Visual Results and Complications Rates in Choyce IOL Implantations: 300 Cases
Indomethacin After ICCE in Prevention of CME: 42 Cases
Combined ECCE with Cyclodialysis: 186 Cases
Endothelial Cell Loss with the Choyce Anterior Chamber Lens
Combined Penetrating Keratoplasty ECCE and IOL Implantation (Triple Procedure): 40 Cases
Indications for Implant Removal
Sterile Hypopyon
Endothelial Cell Loss Following Minimum Trauma Surgery
Iris Erosion in Pediatric Patients
The "Toxic Lens" Syndrome
Frequency of Endophthalmitis with ICCE and Anterior Chamber IOL Results
Post OP Pachymetry and Keratometry as Measures of Healing
IOL Surgery as an Office Procedure

New Designs Reviewed

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Sunday, April 8

Endothelial Photography Course
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William Bourne, M.D.
Miles Galin, M.D.
Herbert Kaufman, M.D.
Manus Kraff, M.D.
Limited attendance 50 $100

Suturing Techniques and Safeguards Course
William Simcoe, M.D., Director
Unlimited attendance Free

Intracapsular Cataract Extraction with Intraocular Lens Course
Norman S. Jaffe, M.D., Director
Henry Clayman, M.D.
Unlimited attendance Free

Extracapsular Cataract Extraction with Intraocular Lens Course
Richard Kratz, M.D., Director
Lawrence Hyde, M.D.
David McIntyre, M.D.
Unlimited attendance Free

Use of McReynolds Instruments Course
William McReynolds, M.D., Director
Ned Snider, M.D.
Unlimited attendance Free

Kelman Anterior Chamber Lens Course
Charles Kelman, M.D., Director
Unlimited attendance Free

The Platina Lens in Intra and Extracapsular Cataract Extraction
Ronald Barnet, M.D., Director
Unlimited attendance Free

Tuesday, April 10

Lens Calculation Course
Kenneth J. Hoffer, M.D., Director
Charles Cleavenger, M.D.
Richard Koplin, M.D.
Lyle Moses, M.D.
H. John Shammas, M.D.
Limited attendance 100 $50

Intracapsular Cataract Extraction with Intraocular Lens Course
Robert C. Drews, M.D., Director
Unlimited attendance Free

Extracapsular Cataract Extraction with Intraocular Lens Course
Henry Hirschman, M.D., Director
David McIntyre, M.D.
William Simcoe, M.D.
Unlimited attendance Free

Malpractice Course
Herve Byron, M.D., Director
Unlimited attendance Free

McCannel Suture Course
Malcolm McCannel, M.D., Director
Unlimited attendance Free

Anterior Chamber Lens Course
Jerald Tennant, M.D., Director
Unlimited attendance Free

Modification of Astigmatism Using Operating Keratometers
Dennis Shepard, M.D., Director
Thomas Cravy, M.D.
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Prior to Mar. 1: ☐ Members, Fellows and Military of AIOIS $325 ☐ Non-Members $400
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☐ Endothelial Photography Course $100 ☐ Ladies' Program $122

After Mar. 1: ☐ Members, Fellows and Military of AIOIS $375 ☐ Non-Members $450
☐ Residents $225 ☐ Nurses and Technicians $150 ☐ Lens Calculation Course $50
☐ Endothelial Photography Course $100 ☐ Ladies' Program $150

☐ Check here if you wish further information on the Ladies' Program

EACH REGISTRANT MUST FILL OUT A SEPARATE FORM. Cancellations: 90% refund prior to postmark date of March 1. 10% refund after postmark date of March 1. Residents must have letter from Chief of department. Make check or money order payable to: U.S. Intraocular Lens Symposium and send with this form to P.O. Box 3140, Santa Monica, California 90403. For hotel reservations, please contact Century Plaza Hotel directly at: Avenue of the Stars, Los Angeles, California 90067. Telephone (213) 277-2000. Toll free reservation number: 1 (800) 228-3000 When making reservations please indicate that you are with the AIOIS.

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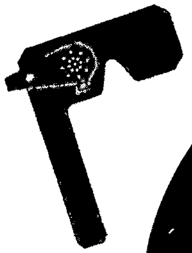

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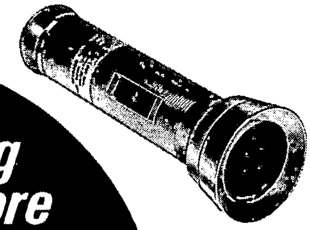

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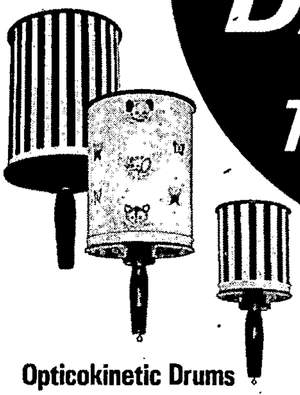

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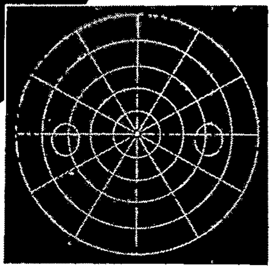

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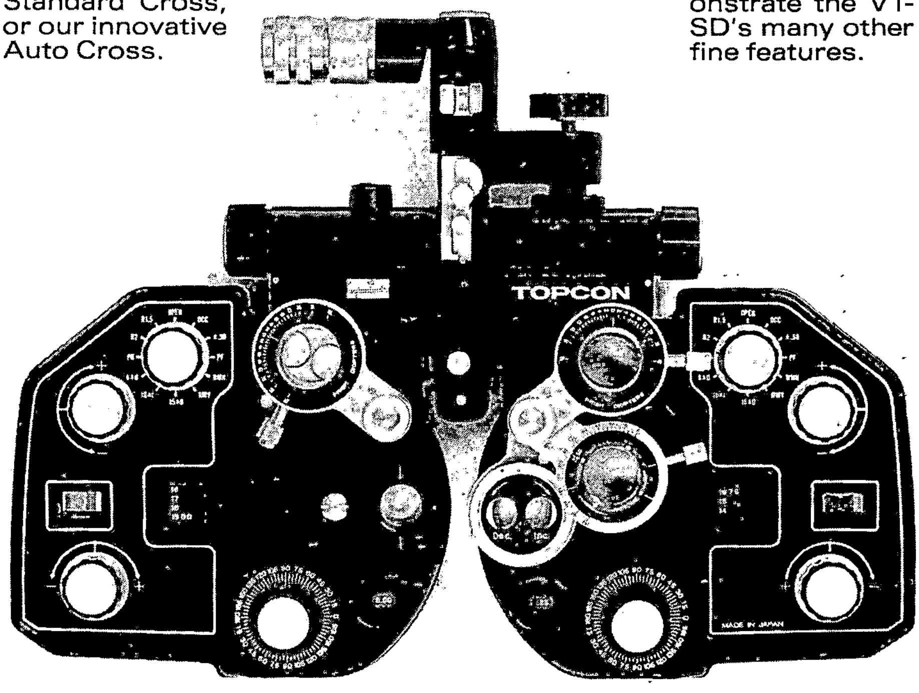

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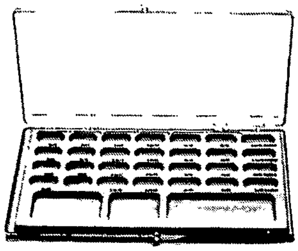

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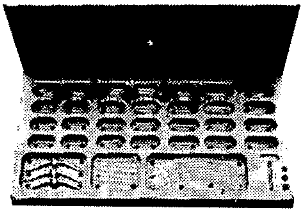

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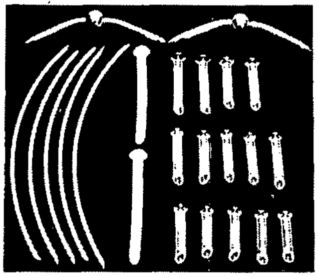

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* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
J.C. Mustarde', L.T. Jones M.D. and A. Callahan M.D., Ophthalmic Plastic Surgery—Up-To-Date, Aesculapius Publishing Company, 1970.
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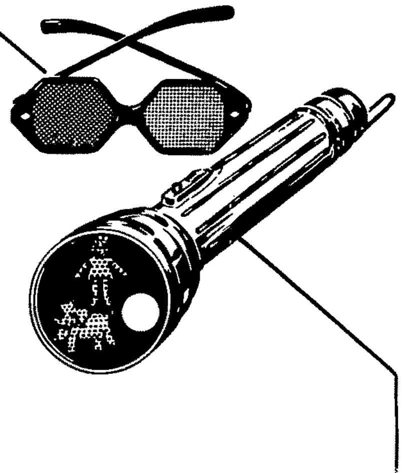

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Mar. 23, 24	University of Texas Medical School San Antonio, TX	David M. Hunter, M.D.	Barbara Woods (512) 696-6295
Apr. 18, 19, 20	University of California Medical School San Francisco, CA	Walter H. Stern, M.D.	Rene Vandergrift (415) 666-4251
May 11, 12	Ingalls Memorial Hospital Harvey, (Chicago) IL	David H. Orth, M.D.	Roberta Martia, R.N. (312) 333-2300, X5811
May 25, 26	Piedmont Hospital Atlanta, GA	William S. Hagler, M.D.	Karen Upchurch (404) 351-2220
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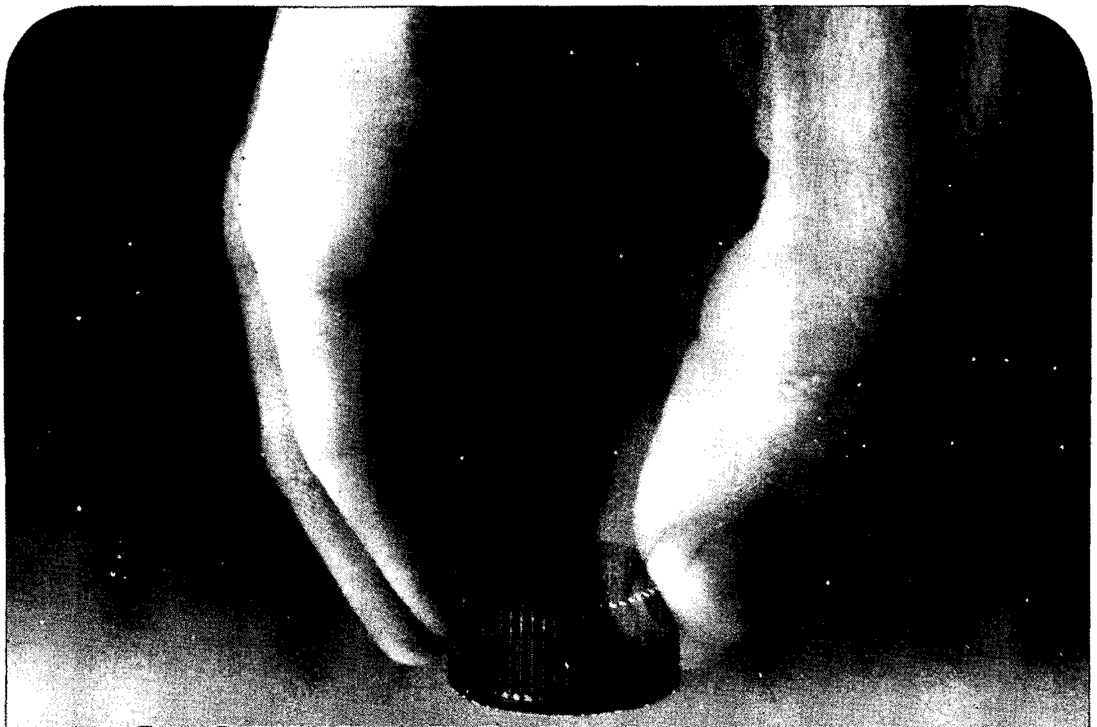

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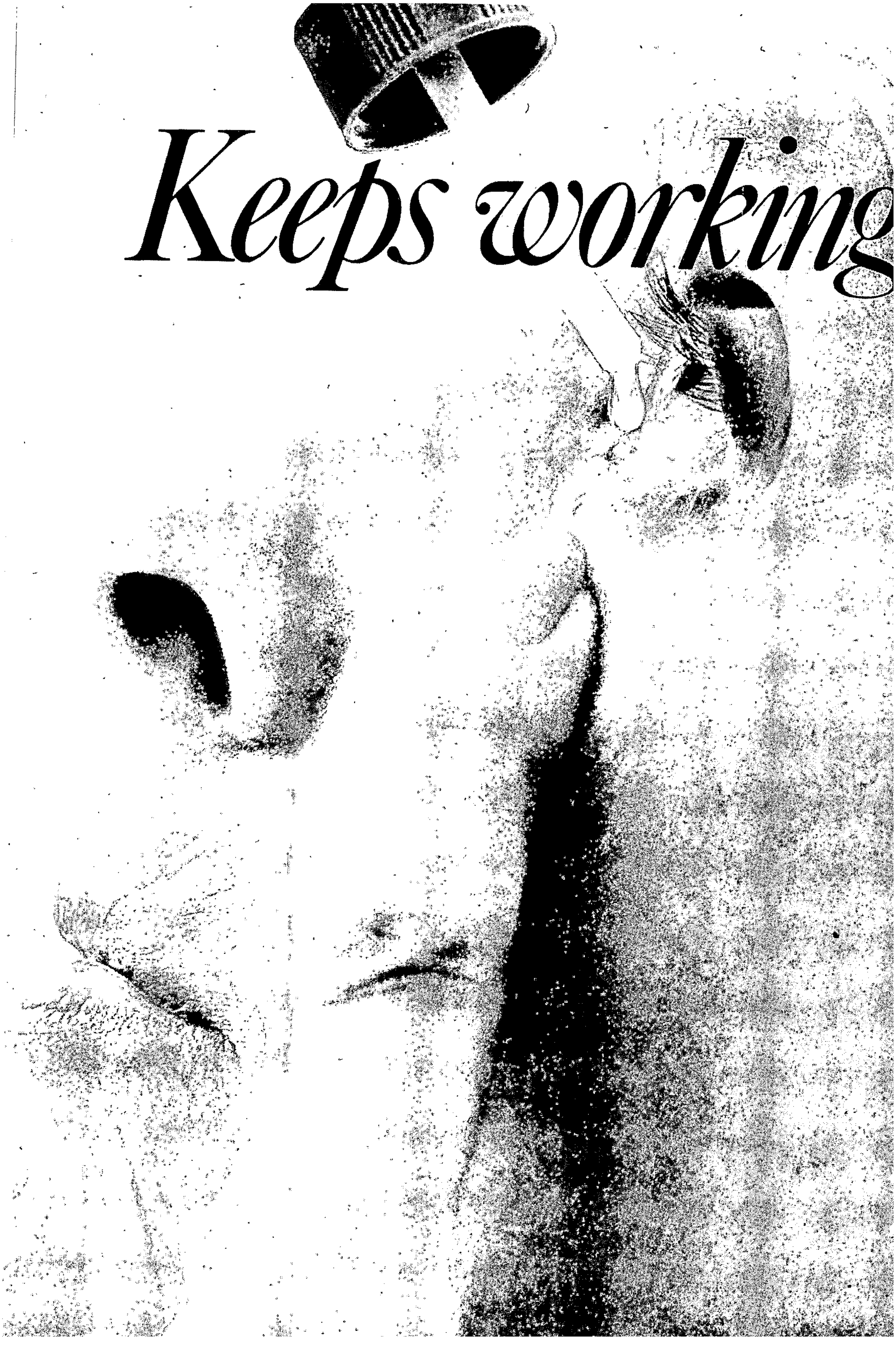
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2. Most cases of angle-closure glaucoma, due to the possibility of increasing angle block.

3. Hypersensitivity to the active or inactive ingredients.

Warnings: 1. *Use in Pregnancy:* Safe use of anticholinesterase medications during pregnancy has not been established, nor has the absence of adverse effects on the fetus or on the respiration of the neonate.

2. Succinylcholine should be administered only with great caution, if at all, prior to or during general anesthesia to patients receiving anticholinesterase medication because of possible respiratory or cardiovascular collapse.

3. Caution should be observed in treating glaucoma with PHOSPHOLINE IODIDE in patients who are at the same time undergoing treatment with systemic anticholinesterase medications for myasthenia gravis, because of possible adverse additive effects.

Precautions: 1. Gonioscopy is recommended prior to initiation of therapy.

2. Where there is a quiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and ciliary muscle contraction that may occur.

3. While systemic effects are infrequent, proper use of the drug requires digital compression of the nasolacrimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

4. Temporary discontinuance of medication is necessary if salivation, urinary incontinence, diarrhea, profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities occur.

5. Patients receiving PHOSPHOLINE IODIDE who are exposed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin. During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable.

6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer, pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.

7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.

8. PHOSPHOLINE IODIDE (echothiophate iodide) should be used with great caution, if at all, where there is a prior history of retinal detachment.

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4. Iris cysts may form, and if treatment is continued, may enlarge and obscure vision. This occurrence is more frequent in children. The cysts usually shrink upon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Rarely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative esotropia.

5. Prolonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.

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Registration fee: Sfrs. 180.— Assistants Sfrs. 90.—

Deadline: February 28, 1979

For information please write to:

UNIVERSITY EYE CLINIC, Prof. Dr. R. Witmer,
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MALIGNANT TUMORS OF THE ORBIT

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under the direction of
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Harvard Faculty:

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January 18 & 19, 1979

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JOSE CUNHA-VAZ, M.D.—University of Illinois Eye & Ear Infirmary, Chicago
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PETER H. FORSHAM, M.D.—University of California, San Francisco
ROBERT L. JACK, M.D.—Palo Alto Retinal Group & Stanford University, Menlo Park & Palo Alto
HOWARD B. LASSMAN, Ph. D.—Somerville, New Jersey
FRANCIS A. L'ESPERANCE, M.D.—Columbia-Presbyterian, New York City
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ARNALL PATZ, M.D.—Johns Hopkins, Baltimore
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FOR REGISTRATION:

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Zweng Memorial Retinal Research Foundation
1225 Crane Street
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Registration Fee: $200 Residents & Fellows: $50 with letter from Dept. Chairman

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Department of Continuing Education
ANNOUNCES A COURSE IN NEURO-OPHTHALMOLOGY
April 9-13, 1979
AT THE HYATT REGENCY HOTEL, CAMBRIDGE, MASS. UNDER THE DIRECTION OF
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OF THE MASSACHUSETTS EYE AND EAR INFIRMARY

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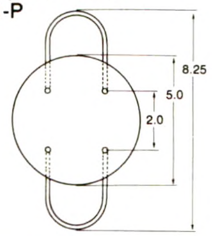

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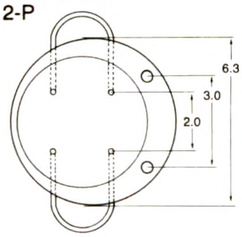

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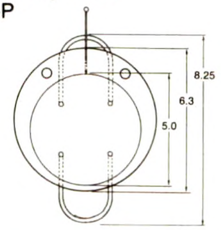

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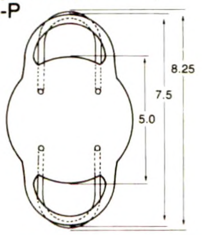

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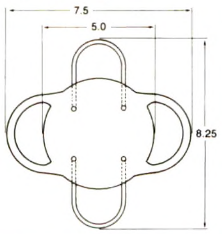

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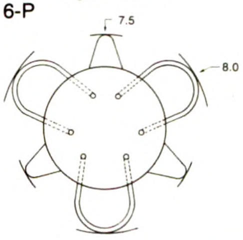

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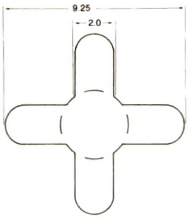
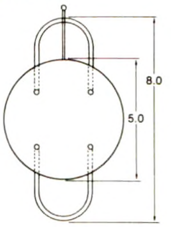


5mm Clip Lens
Style 16



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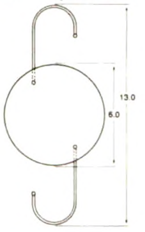

Anterior Chamber Lens
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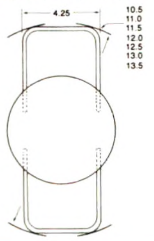

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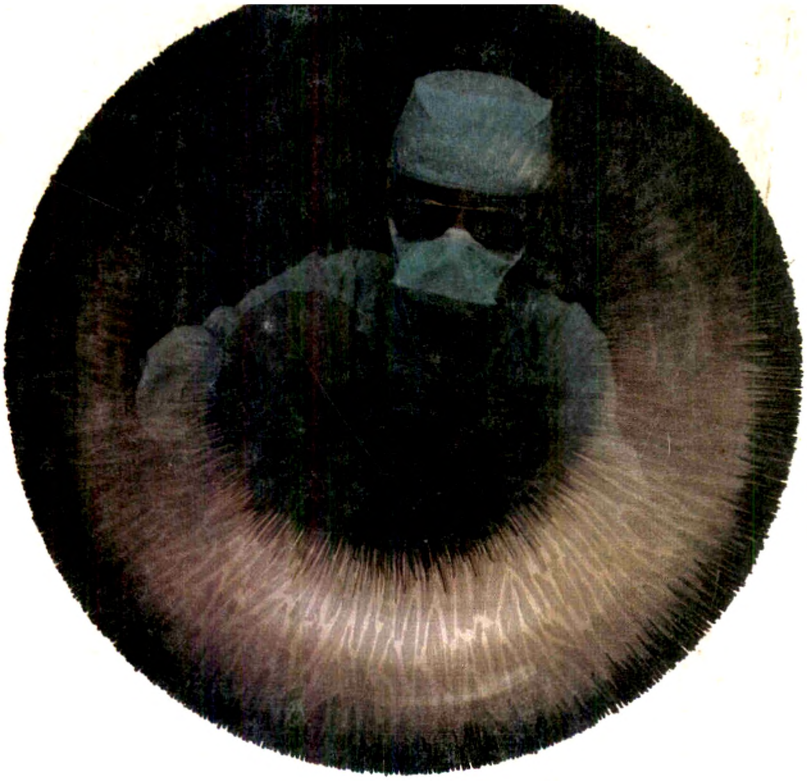

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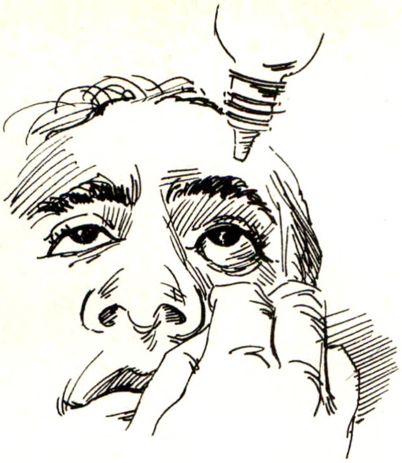

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Isopto Carpine has a 25-year history of proven effectiveness in the treatment of glaucoma. Its abilities to reduce intraocular pressure[1], to prolong corneal contact time and to lubricate and soothe[2] have been noted repeatedly in clinical studies. Isopto Carpine is made by Alcon, a name synonymous with consistent quality in ophthalmic products, and is widely available in eleven strengths from 0.25 to 10%. Patients with limited means may obtain Isopto Carpine without cost through the Alcon Needy Patient Assistance Program.

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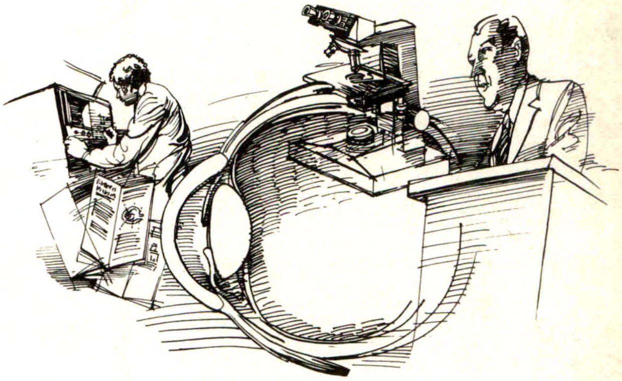
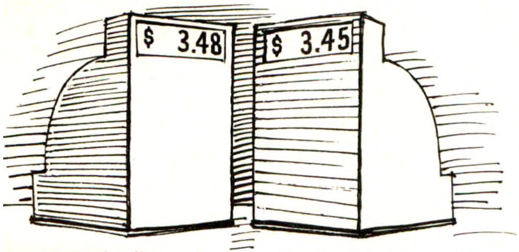
Your prescription for Isopto Carpine helps further Alcon's continuing commitment to ophthalmic research (over $9 million expended since 1973). It also enables us to provide drug grants, speaker grants, patient information aids and educational materials on glaucoma, to support national and local ophthalmology societies, and to make free medication available to indigent patients.





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Some pharmaceuticals may be substantially cheaper when prescribed generically, but surprisingly, not Isopto Carpine. A recent nationwide survey of drugstores and pharmacies[3] has again demonstrated that on the average, the actual cost to the patient of Isopto Carpine is usually no more (give or take pennies) and often actually less than generic pilocarpine, ml for ml.

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(Pilocarpine HCl)

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1. Haas, J. S. and Merrill, D. L.: Am. J. Ophthal. *54*:1, 1962.
2. Linn, M. L. and Jones, L. T.: Am. J. Ophthal. *65*:1, 1968.
3. Independent Prescription Audit. Data on file at Alcon Laboratories.

Description: A sterile ophthalmic solution. Each ml contains: Active: Pilocarpine Hydrochloride 0.25%, 0.5%, 1%, 1.5%, 2%, 3%, 4%, 5%, 6%, 8%, or 10%. Preservative: Benzalkonium Chloride 0.01%. Vehicle: Hydroxypropyl Methylcellulose 0.5%. Inactive: Boric Acid, Sodium Chloride (present in 0.25%, 0.5%, and 1% only), Sodium Citrate, Hydrochloric Acid and/or Sodium Hydroxide (to adjust pH in 1%, 1.5%, 2%, 3%, 4%, 5%, 6%, 8%, and 10%). Citric Acid, Hydrochloric Acid and/or Sodium Hydroxide (to adjust pH in 0.25% and 0.5%). Purified Water. **Contraindications:** When constriction is undesirable such as in acute iritis and in cases of hypersensitivity to any of the components. **Precaution:** Avoid overdosage. **Adverse Reactions:** Slight ciliary spasm with temporary reduction in visual acuity and headaches. Contact allergy may occur with prolonged use. Sensitivity is infrequently observed. Retinal detachment has been reported. Lens opacities may occur with prolonged use. Systemic reactions are rare. **Dosage:** 2 drops in the eye(s) 3 to 4 times daily. **How Supplied:** 15ml and 30ml Drop-Tainer® dispensers.

VIRA-A®
(vidarabine ophthalmic ointment), 3%

Description. Vira-A (vidarabine, adenine arabinoside, or Ara-A) is an antiviral drug for topical ophthalmic administration. The chemical name is 9-β-D-arabinofuranosyladenine. Each gram of the ophthalmic ointment contains 30 mg of vidarabine monohydrate equivalent to 28.11 mg of vidarabine in a sterile, inert, petrolatum base.

Clinical Pharmacology. Vira-A is a purine nucleoside obtained from fermentation cultures of *Streptomyces antibioticus.* Vira-A possesses *in vitro* and *in vivo* antiviral activity against Herpes simplex types 1 and 2, Varicella-Zoster, and Vaccinia viruses. Except for Rhabdovirus and Oncornavirus, Vira-A does not display *in vitro* antiviral activity against other RNA or DNA viruses, including Adenovirus.

The antiviral mechanism of action has not been established. Vira-A appears to interfere with the early steps of viral DNA synthesis. Vira-A is rapidly deaminated to arabinosylhypoxanthine (Ara-Hx), the principal metabolite. Ara-Hx also possesses *in vitro* antiviral activity but this activity is less than that of Vira-A. Because of the low solubility of Vira-A, trace amounts of both Vira-A and Ara-Hx can be detected in the aqueous humor only if there is an epithelial defect in the cornea. If the cornea is normal, only trace amounts of Ara-Hx can be recovered from the aqueous humor.

Systemic absorption of Vira-A should not be expected to occur following ocular administration and swallowing lacrimal secretions. In laboratory animals, Vira-A is rapidly deaminated in the gastrointestinal tract to Ara-Hx.

In contrast to topical idoxuridine, Vira-A demonstrated less cellular toxicity in the regenerating corneal epithelium of the rabbit.

Indications and Usage. Vira-A Ophthalmic Ointment, 3%, is indicated for the treatment of acute keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus types 1 and 2. Vira-A is also effective in superficial keratitis caused by Herpes simplex virus which has not responded to topical idoxuridine or when toxic or hypersensitivity reactions to idoxuridine have occurred. The effectiveness of Vira-A Ophthalmic Ointment, 3%, against stromal keratitis and uveitis due to Herpes simplex virus has not been established.

The clinical diagnosis of keratitis caused by Herpes simplex virus is usually established by the presence of typical dendritic or geographic lesions on slit-lamp examination. In controlled and uncontrolled clinical trials, an average of seven and nine days of continuous Vira-A Ophthalmic Ointment, 3%, therapy was required to achieve corneal re-epithelialization. In the controlled trials, 70 of 81 subjects (86%) re-epithelialized at the end of three weeks of therapy. In the uncontrolled trials, 101 of 142 subjects (71%) re-epithelialized at the end of three weeks. Seventy-five percent of the subjects in these uncontrolled trials had either not healed previously or had developed hypersensitivity to topical idoxuridine therapy.

The following topical antibiotics: gentamicin, erythromycin, chloramphenicol; or topical steroids: prednisolone or dexamethasone, have been administered concurrently with Vira-A Ophthalmic Ointment, 3%, without an increase in adverse reactions.

Normally, corticosteroids alone are contraindicated in Herpes simplex virus infections of the eye. If Vira-A Ophthalmic Ointment, 3%, is administered concurrently with topical corticosteroid therapy, corticosteroid-induced ocular side effects must be considered. These include corticosteroid-induced glaucoma or cataract formation and progression of a bacterial or viral infection.

Vira-A is not effective against RNA virus or adenoviral ocular infections. Vira-A is also not effective against bacterial, fungal, or chlamydial infections of the cornea or nonviral trophic ulcers.

Contraindication. Vira-A Ophthalmic Ointment, 3%, is contraindicated in patients who develop hypersensitivity reactions to it.

Warnings. ***Use in Pregnancy:*** Vira-A (vidarabine) parenterally is teratogenic in rats and rabbits. Ten percent Vira-A ointment applied to 10% of the body surface during organogenesis induced fetal abnormalities in rabbits. When 10% Vira-A ointment was applied to 2 to 3% of the body surface of rabbits, no fetal abnormalities were found. This dose greatly exceeds the total recommended ophthalmic dose in humans. The possibility of embryonic or fetal damage in pregnant women receiving Vira-A (vidarabine ophthalmic ointment), 3%, is remote. The topical ophthalmic dose is small, and the drug relatively insoluble. Its ocular penetration is very low. However, a safe dose for a human embryo or fetus has not been established. Consequently, Vira-A should be used only when clearly indicated.

It is not known whether Vira-A is excreted in human milk. As a general rule, because many drugs are excreted in human milk, nursing should not be undertaken while a patient is under treatment. However, excretion of vidarabine in breast milk is unlikely because Vira-A is rapidly deaminated in the gastrointestinal tract.

Mutagenic Potential: Results of *in vitro* experiments indicate that vidarabine can be incorporated into mammalian DNA and can induce mutation in mammalian cells (mouse L5178Y cell line). Thus far, *in vivo* studies have not been as conclusive, but there is some evidence (dominant lethal assay in mice) that vidarabine may be capable of producing mutagenic effects in male germ cells.

It has also been reported that vidarabine causes chromosome breaks and gaps when added to human leukocytes *in vitro.* While the significance of these effects in terms of mutagenicity is not fully understood, there is a well-known correlation between the ability of various agents to produce such effects and their ability to produce heritable genetic damage.

Oncogenic Potential: Chronic parenteral (IM) studies of vidarabine have been conducted in mice and rats.

In the mouse study, there was a statistically significant increase in liver tumor incidence among the vidarabine-treated females. In the same study, some vidarabine-treated male mice developed kidney neoplasia. No renal tumors were found in the vehicle-treated control mice or the vidarabine-treated female mice.

In the rat study, intestinal, testicular, and thyroid neoplasia occurred with greater frequency among the vidarabine-treated animals than in the vehicle-treated controls. The increases in thyroid adenoma incidence in the high-dose (50 mg/kg) males and the low-dose (30 mg/kg) females were statistically significant.

Hepatic megalocytosis, associated with vidarabine treatment, has been found in short- and long-term rodent (rat and mouse) studies. It is not clear whether or not this represents a preneoplastic change.

The recommended frequency and duration of administration should not be exceeded (see Dosage and Administration).

Precautions. The diagnosis of keratoconjunctivitis due to Herpes simplex virus should be established clinically prior to prescribing Vira-A Ophthalmic Ointment, 3%.

Patients should be forewarned that Vira-A Ophthalmic Ointment, 3%, like any ophthalmic ointment, may produce a temporary visual haze.

Although viral resistance to Vira-A has not been observed, this possibility may exist.

Adverse Reactions. Lacrimation, foreign-body sensation, conjunctival injection, burning, irritation, superficial punctate keratitis, pain, photophobia, punctal occlusion, and sensitivity have been reported with Vira-A Ophthalmic Ointment, 3%. The following have also been reported but appear disease-related: uveitis, stromal edema, secondary glaucoma, trophic defects, corneal vascularization, and hyphema.

Overdosage. Acute massive overdosage by oral ingestion of the ophthalmic ointment has not occurred. However, the rapid deamination to arabinosylhypoxanthine should preclude any difficulty. The oral LD_{50} for vidarabine is greater than 5,020 mg/kg in mice and rats. No untoward effects should result from ingestion of the entire contents of a tube.

Overdosage by ocular instillation is unlikely because any excess should be quickly expelled from the conjunctival sac. Too frequent administration should be avoided.

Dosage and Administration. Administer approximately one half inch of Vira-A Ophthalmic Ointment, 3%, into the lower conjunctival sac five times daily at three-hour intervals.

If there are no signs of improvement after 7 days, or complete re-epithelialization has not occurred by 21 days, other forms of therapy should be considered. Some severe cases may require longer treatment.

After re-epithelialization has occurred, treatment for an additional seven days at a reduced dosage (such as twice daily) is recommended in order to prevent recurrence.

How Supplied. N 0071-3677-07 (Stock 18-1677-139)

Vira-A Ophthalmic Ointment, 3%, is supplied sterile in ophthalmic ointment tubes of 3.5 g. The base is a 60:40 mixture of solid and liquid petrolatum. **UK**

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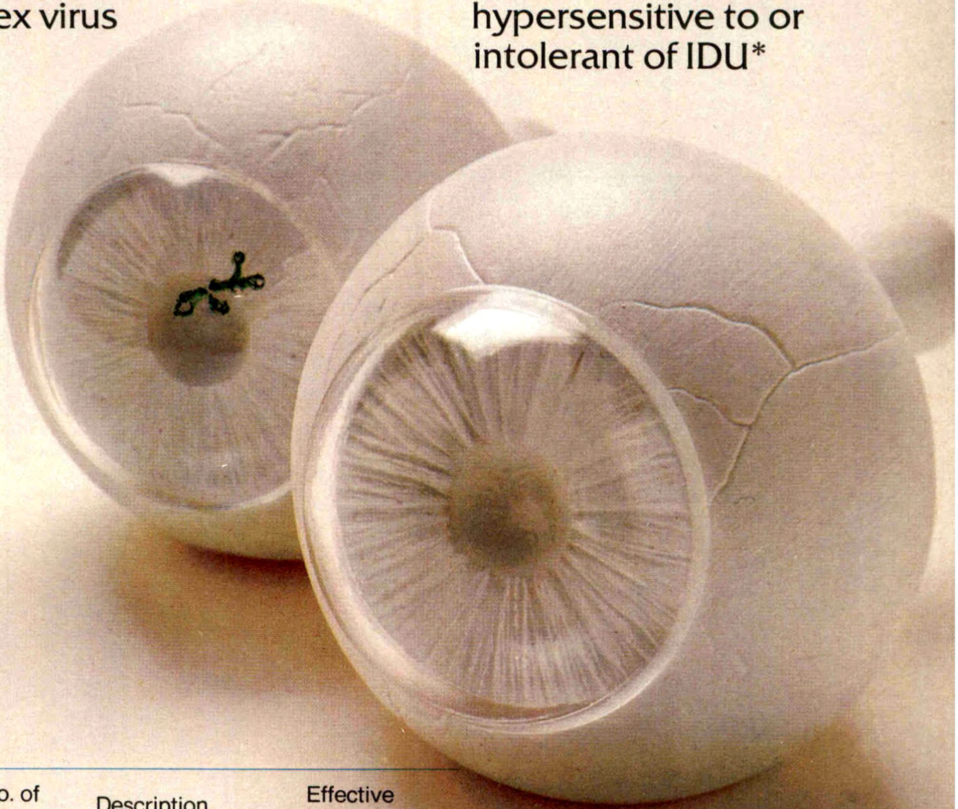

Clinical Evidence	No. of Subjects	Description	Effective Response
Vira-A Controlled Trials	81	Previously untreated	70 subjects **(86%)**
Vira-A Uncontrolled Trials	142	75% had been resistant or hypersensitive to IDU	101 subjects **(71%)**

NOTE: In the controlled and uncontrolled trials, 70 and 101 subjects, respectively, re-epithelialized at the end of three weeks on Vira-A.

Vira-A is preservative-free.

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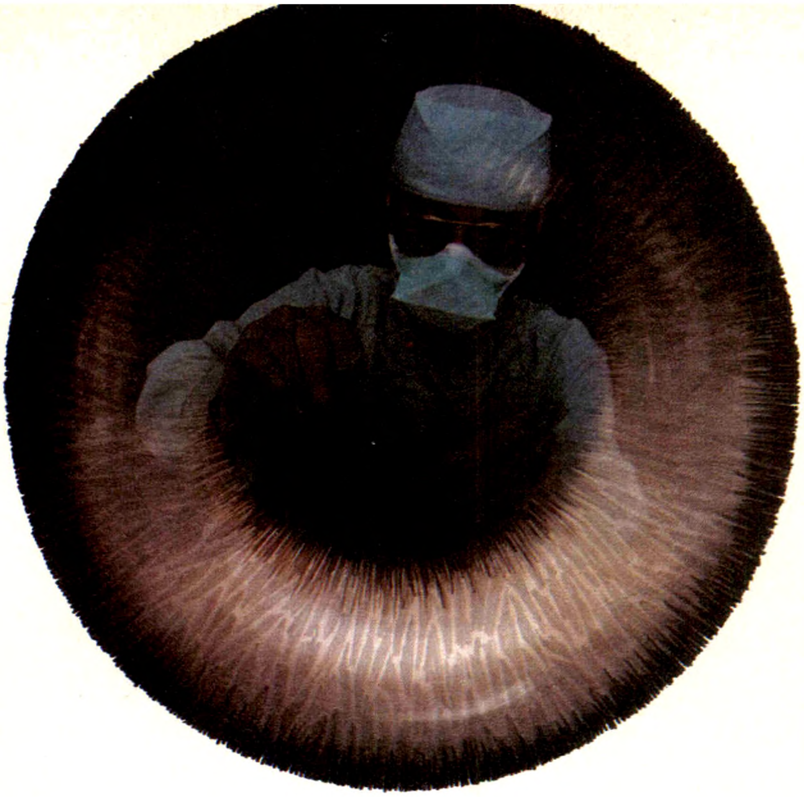

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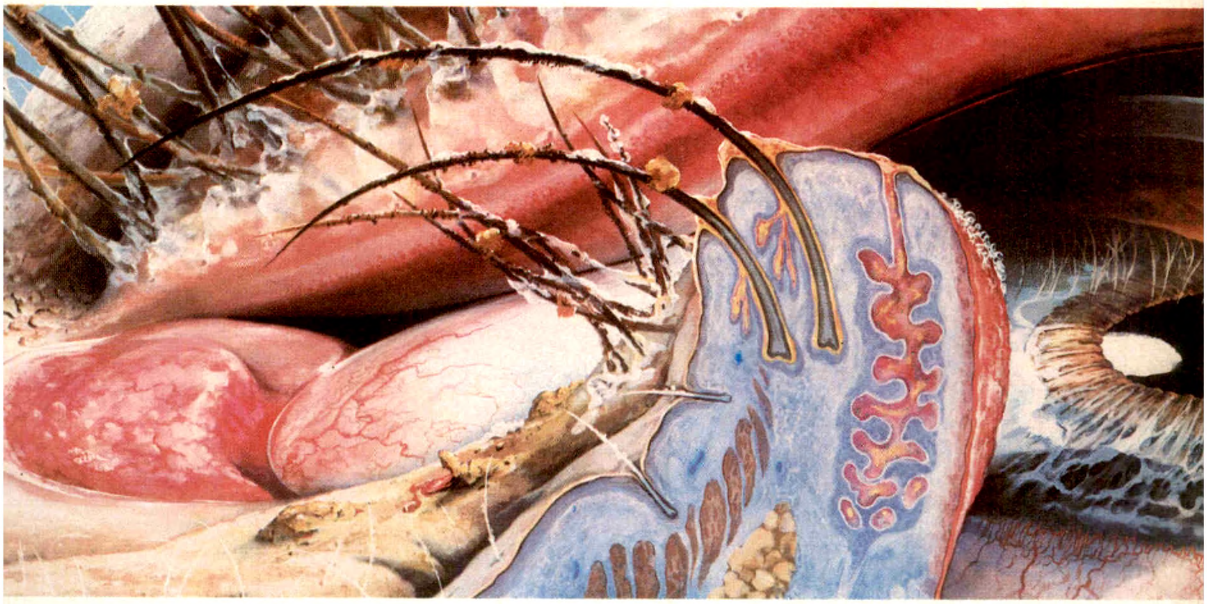
Blepharitis*: A complex subject.



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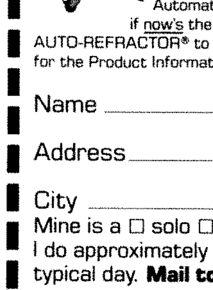

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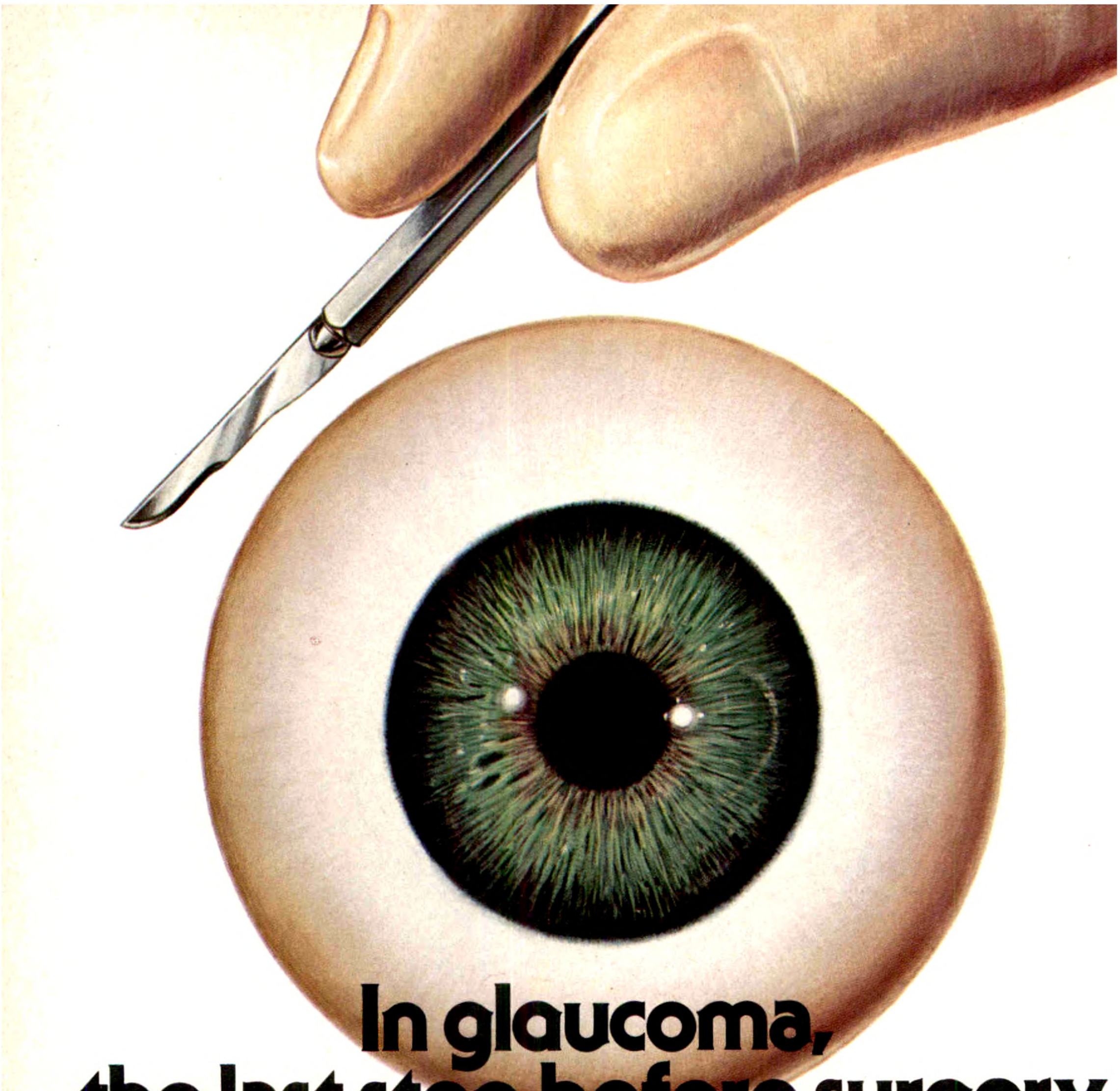

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Contraindications: Severe or absolute glaucoma and chronic noncongestive angle closure glaucoma. Of doubtful use in glaucoma due to severe peripheral anterior synechiae or hemorrhagic glaucoma. Adrenocortical, hepatic, or renal insufficiency; electrolyte imbalance state, e.g., hyperchloremic acidosis; sodium and potassium depletion states.

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[1] Havener, W.H., Ocular Pharmacology, Third Ed., The C. V. Mosby Co., St. Louis, 1974, pp. 478

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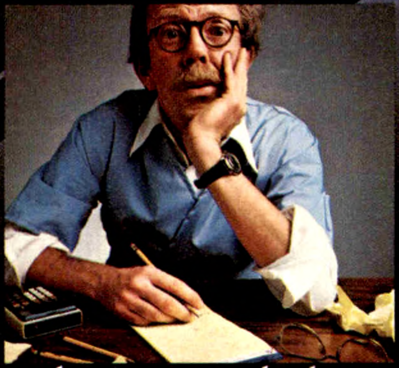

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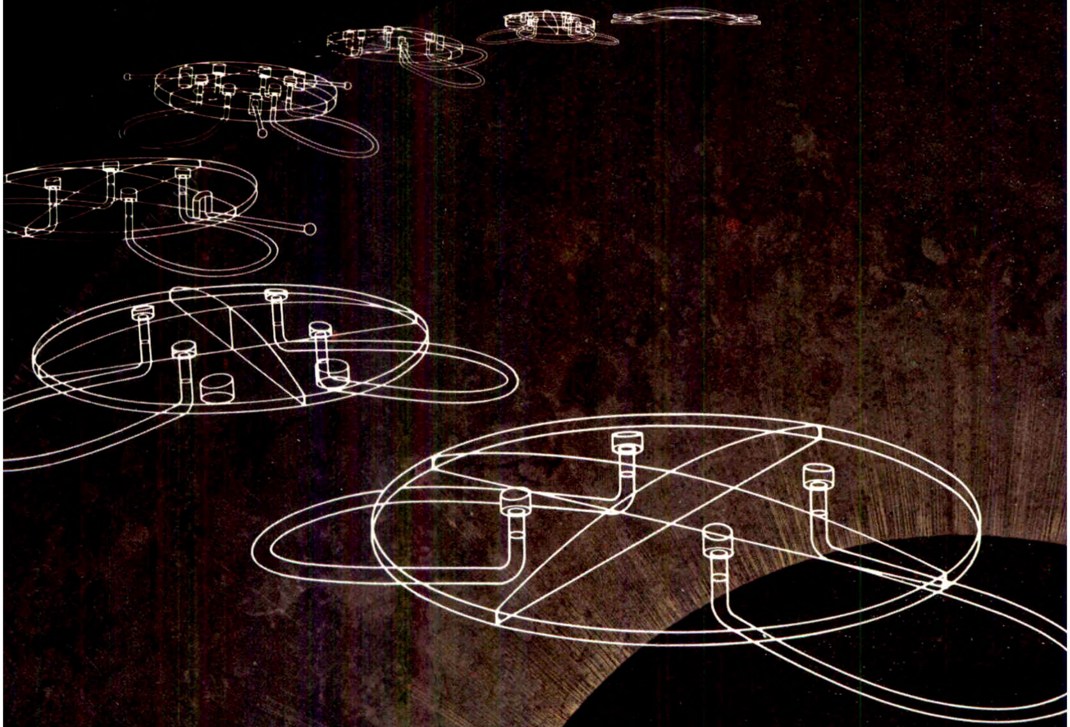

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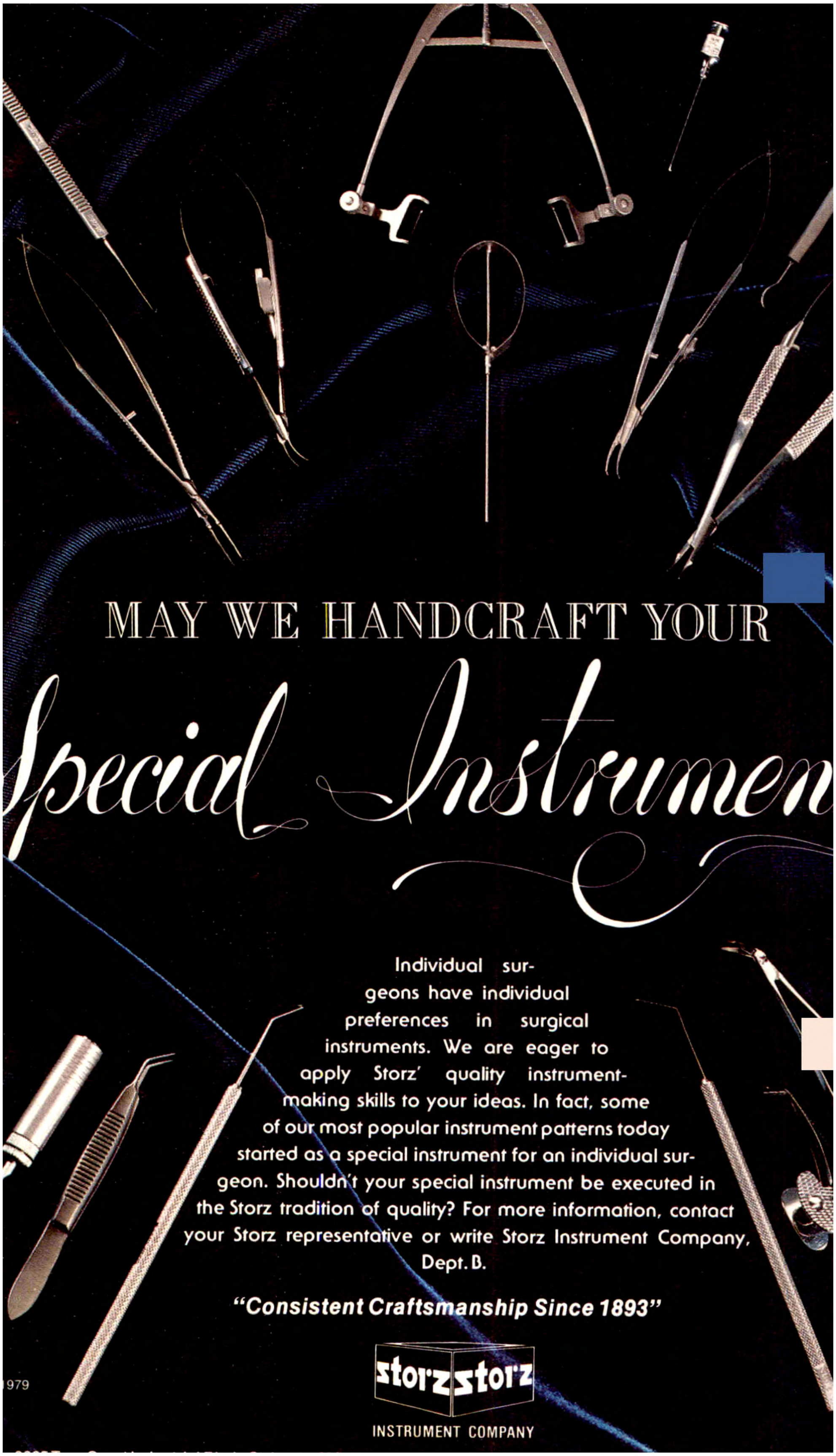

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See full prescribing information which follows on last page

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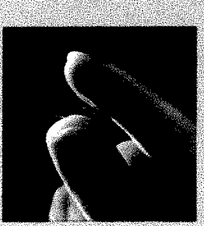

BAUSCH & LOMB SOFLENS® (polymacon) Contact Lenses

VISION CORRECTION USE

DESCRIPTION: The SOFLENS® (polymacon) Contact Lens is a hemispherical flexible shell which covers the cornea and may cover a portion of the adjacent sclera. It consists of 61.4% poly (2-hydroxy-ethyl methacrylate) and 38.6% water by weight when immersed in normal saline. The material has a refractive index of 1.43 and the lens has a visible light transmittance of more than 97%.

ACTIONS: In its hydrated state a SOFLENS®(polymacon) Contact Lens is soft and pliable. When placed on the human cornea, the hydrated SOFLENS®(polymacon)Contact Lens acts as a refracting medium to compensate spherical ametropias.

INDICATIONS: SOFLENS® (polymacon) Contact Lenses are indicated for vision correction use in persons with non-diseased eyes and in aphakia.

CONTRAINDICATIONS: SOFLENS® (polymacon) Contact Lenses are contraindicated in the presence of any active untreated infection or abnormality of the anterior segment of the eye with the exception of ametropia and aphakia.

WARNINGS: Abrasions and Infections—If a lens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present, or any abnormal eye condition is observed concurrent with lens wear, the lens should be removed immediately and a physician consulted.

Infectious corneal ulcers have been reported, usually associated with failure to follow the recommended procedures for care of the lenses.

Aphakic Patients—Aphakic patients should not be fitted with SOFLENS®(polymacon) Contact Lenses during the postoperative period until, in the opinion of the surgeon, the eye has healed completely.

Lens Sanitation and Handling—Persons who require only vision correction and who would not, or could not, adhere to the recommended daily sanitary care of SOFLENS®(polymacon) Contact Lenses or who are unable to place and remove the lenses should not be provided with them. Failure to follow handling and sanitation instructions could lead to serious eye infections which might result in corneal ulcers.

Malfunction and rusting of the metal interior of the BAUSCH & LOMB™ Disinfecting Unit (ASEPTOR®) as well as discoloration and cracking of the lens case has been reported after varying periods of use. If such occurs, appropriate replacement is indicated to avoid interference with the disinfection procedure. Keep the BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) clean and dry at all times.

Medicants and Eye Drops—When the lenses are used by persons requiring only vision correction no ophthalmic solutions or medicants, including conventional (hard) contact lens solutions and eye drops, except BAUSCH & LOMB™ Lens Lubricant, should be used by SOFLENS® (polymacon) Contact Lens wearers prior to placement, or while the lens is in place, on the eye. Also, no solutions, including conventional (hard) contact lens solutions other than daily cleaner, lens lubricant, saline, and the solution made from the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets are to be used on a SOFLENS® (polymacon) Contact Lens when the lens is off the eye.

Wearing Restrictions—SOFLENS® (polymacon) Contact Lenses when used only for vision correction should be removed before sleeping or swimming and in the presence of noxious and irritating vapors. Insufficient lacrimal secretions or hypoesthesia may be restricting factors to the wearing of soft contact lenses.

PRECAUTIONS: Storage—SOFLENS® (polymacon) Contact Lenses must be stored ONLY in saline solution. If left exposed to air, the lenses will dehydrate, become brittle, and break readily. If a lens dehydrates, it should be resoaked in saline solution until it returns to its soft, pliable state which may take as long as forty minutes.

Fresh saline solution must be used DAILY for cleaning, storing, and disinfection of lenses. The carrying case must be emptied and refilled with fresh saline solution just before disinfecting the lenses.

If using BAUSCH & LOMB™ Salt Tablets to make saline solution, the solution **MUST BE PREPARED FRESH DAILY.** As this saline is not aseptic, it should not be used to clean a lens that is to be replaced immediately on the eye, nor should this solution ever be placed in the eye. If the patient wishes to remove a lens, clean it, or wet it and replace it on the eye, the solution in the carrying case should be used, as it will have been disinfected, or Preserved Saline Solution may be used.

Hygiene—Hands must be washed, rinsed thoroughly, and dried with a lint-free towel before handling the lenses.

Cosmetics, lotions, soaps, and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the hair spray has settled.

Handling—A SOFLENS®(polymacon) Contact Lens may be damaged by nicking or tearing if care is not exercised during placement on or removal from the eye, replacing or removing it from the carrying case or in the cleaning process. Lenses must be placed very carefully in the carrying case to avoid damaging the edges of the lenses.

Disinfecting—Fresh saline solution must be used DAILY for cleaning, storing and disinfection of lenses. If using BAUSCH & LOMB™ Salt Tablets to make saline solution, the solution MUST BE PREPARED FRESH DAILY. After removal from the eye, the SOFLENS® (polymacon) Contact Lens must be rubbed gently with Daily Cleaner then rinsed with saline solution to remove mucus and film from the lens surface. The BAUSCH & LOMB™ Lens Carrying Case (LENSGARD™) must be emptied and refilled with fresh saline solution just before disinfecting the lenses.

The causes and nature of deposits formed on the surfaces of some lenses have not been completely evaluated. However, some coatings are known to be proteinaceous and others may be oily or greasy film from extraneous agents, such as hair spray or other cosmetics, or from the patient's own lacrimal secretions. Many wearers experience little or no difficulty with such deposits. However, occasionally a wearer, who tends to secrete unusually large amounts of mucus in the lacrimal fluid, may experience a build-up of these deposits within a relatively few weeks, despite adequate cleaning measures. If surface accumulations of non-removable materials persist, professional care should be obtained promptly.

Deposits, characterized as medium or heavy, were found on 17.5 percent of a population of lenses worn an average of 11.2 months, including lenses worn as long as thirty months. The occurrence of these deposits appeared to increase with the duration of lens use. These medium or heavy deposits, when they do exist, can be detected by means of a slit lamp biomicroscope examination. Light deposits, unrelated to length of lens use and of no apparent clinical significance, were observed on approximately one-half of the lenses studied.

The BAUSCH & LOMB™ Lens Carrying Case (LENSGARD™) should be washed at least once a week with hot water and then rinsed thorougily with saline solution. Soap or other cleaners should never be used to clean the carrying case.

In order to remove protein deposits which may form on the lenses, wearers should use the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets according to the directions for use which accompany the tablets. To help remove tear residues and mucus as well as other deposits that tend to accumulate on the lens surface, patients should use Daily Cleaner according to the directions for use which accompany the product.

Disinfecting with a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is necessary to kill microorganisms.

If a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is not available, the lenses may be disinfected by boiling them in their carrying case in a pan of water for ten minutes. When this boiling method of disinfection is used, the lenses can be damaged if the boiling water is allowed to completely evaporate.

The BAUSCH & LOMB™ Lens Carrying Case (LENSGARD™) must **always be 2/3 full** of fresh saline solution before disinfecting and must **always** be tightly closed to prevent leakage of the saline from the case and subsequent dehydration of the lenses.

Fluorescein—Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with sterile normal saline solution and wait at least one hour before replacing the lenses. Earlier replacement may cause the lenses to absorb residual fluorescein.

ADVERSE REACTIONS: Serious corneal damage may result from wearing a SOFLENS® (polymacon) Contact Lens which has been soaked in a conventional contact lens solution.

Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritation.

A lens adheres very rarely to an eye as a result of sleeping with the lens on, or wearing a hypotonic lens. If a lens adheres for any reason, apply fresh saline and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema, as manifested by symptoms such as rainbows, halos around lights, or foggy vision, may occur in less than 5% of SOFLENS® (polymacon) Contact Lens wearers. If these symptoms occur, the lenses should be removed and professional consultation obtained.

Excessive tearing, unusual eye secretions, and photophobia are not normal. If these symptoms occur, the patient should be examined to determine their cause.

A faint blue haze, believed to be located in Descemet's membrane, has been reported in the Spokane, Washington area. As yet, the cause is unknown and the phenomenon has not been found elsewhere. The wearers report no subjective symptoms and there is no detectable effect on their visual acuity. There have been approximately 29 cases, and in 10 of these patients the blue haze has cleared or is in various stages of regression.

Neovascularization of the cornea has been observed in some aphakic patients fitted with the SOFLENS®(polymacon) Contact Lens, which may require discontinuation of the lens. Medical consultation should be obtained in such an instance.

LENS REPLACEMENT: Various studies have been conducted to determine the frequency of lens replacements and the reasons for those replacements. These studies show that during the first four months of wear, approximately one quarter of the lenses initially dispensed are replaced; the principal reason for these replacements is lens damage. Lenses are more apt to be damaged while new wearers are learning the prescribed handling and care techniques. After the initial four months of wear, the average lens replacement rate has been found to be approximately one lens per wearer per year. Lens loss, damage and surface deposits were the major causes for lens replacements.

DOSAGE AND ADMINISTRATION: Conventional methods of fitting contact lenses do not apply to SOFLENS®(polymacon) Contact Lenses. For a detailed description of the fitting technique, refer to the SOFLENS Fitting Guide, copies of which are available from: SOFLENS Division, Bausch & Lomb Incorporated, Rochester, N.Y. 14602.

When the lenses are used only for vision correction, there may be a tendency for the patient to overwear the lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to these patients:

Day	Wear Time (hours)	Rest Period (hours)	Wear Time (hours)	Rest Period (hours)	Wear Time (hours)
1	3	1	3	1	3
2	3	1	3	1	3
3	4	1	4	1	4
4	4	1	4	1	4
5	6	1	6	1	4
6	6	1	6	1	4
7	8	1	8		
8	8	1	8		
9	8	1	8		
10	10	1	balance of the waking hours*		
11	12	1	balance of the waking hours*		
12	14	1	balance of the waking hours*		

*Lenses used only for vision correction should never be worn 24 hours a day.

When lenses are dispensed for vision correction, the wearer must be supplied with a lens care kit and must fully understand all lens care and handling instructions. As with any contact lens, regular recall visits are necessary to assure corneal health and wearer compliance with instructions.

HOW SUPPLIED: Each lens is supplied sterile in a glass vial containing sterile normal saline solution. The glass vial is marked with the manufacturing lot number of the lens and the dioptric power (black for plus power lenses; red for minus; gold or white for plano).

Caution: Federal Law Prohibits Dispensing Without a Prescription.

A BAUSCH & LOMB™ Care Kit is available for lens disinfecting, cleaning, and storage.

Complete information on lenses and accessory products can be found in the current SOFLENS Fitting Guide, price list, or sales policy.

SEPTEMBER 1978

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SOFLENS DIVISION
Rochester, N.Y. 14602

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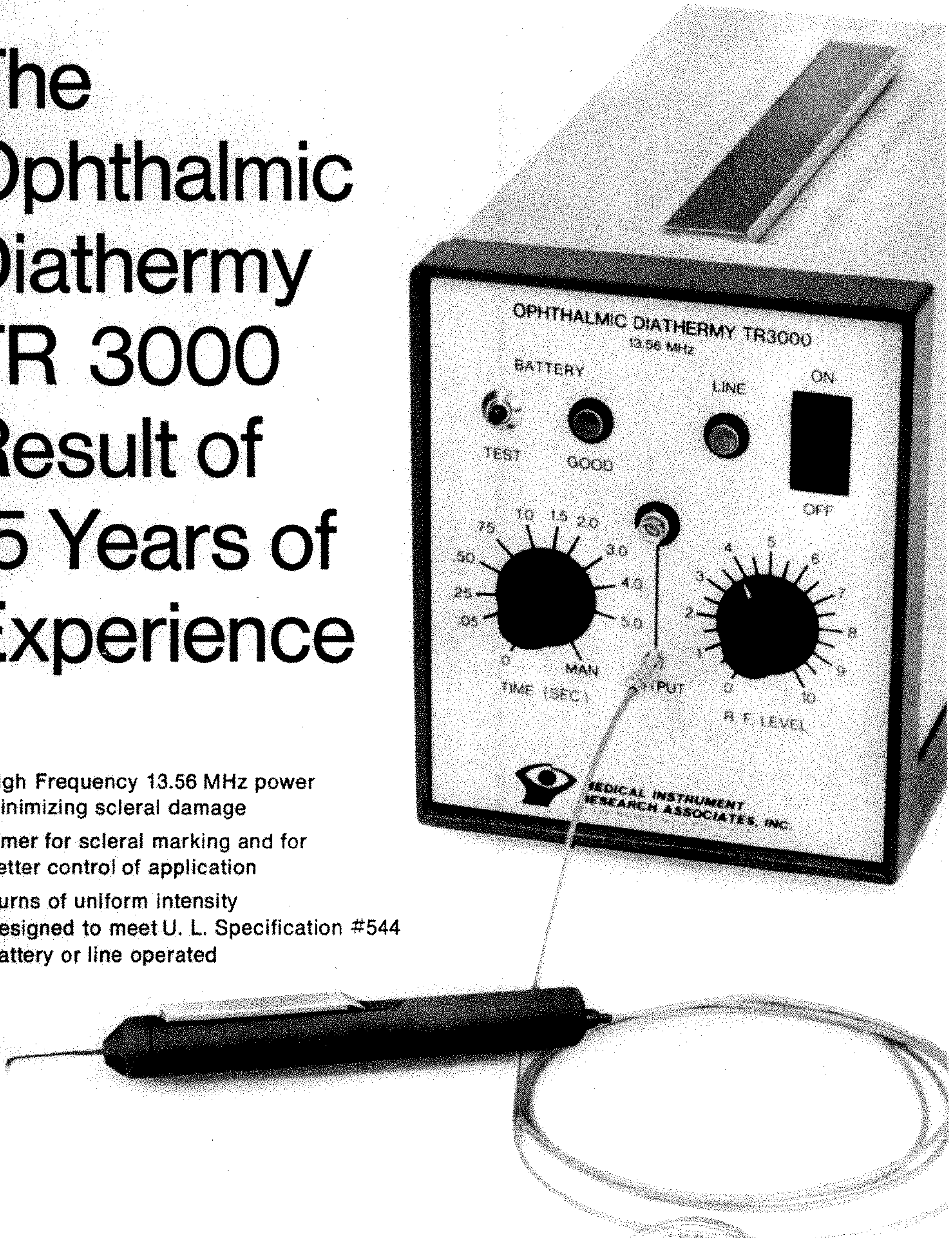

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- **Isotonic formulation helps maintain normal corneal thickness.** To help maintain optimum ocular clarity and comfort, and to help avoid corneal swelling or thinning, Visalens is specially formulated to the tonicity of natural tears.
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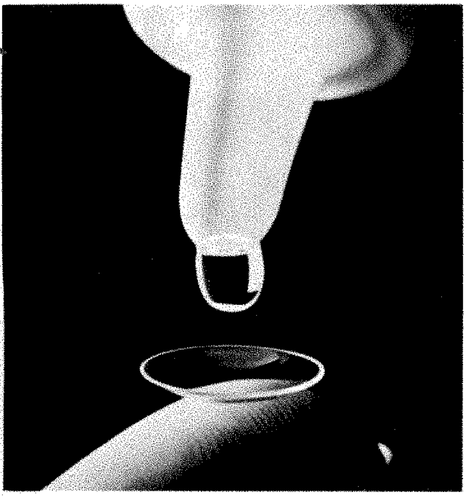

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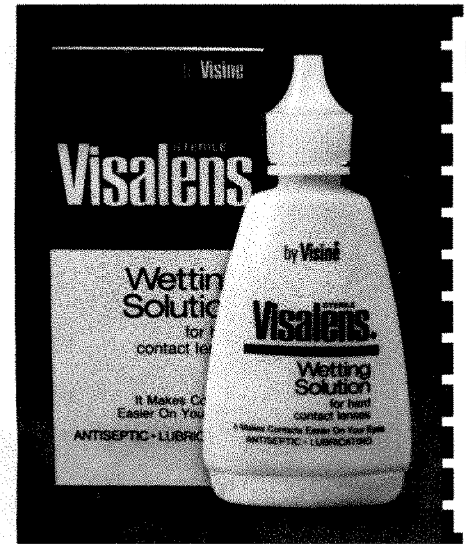

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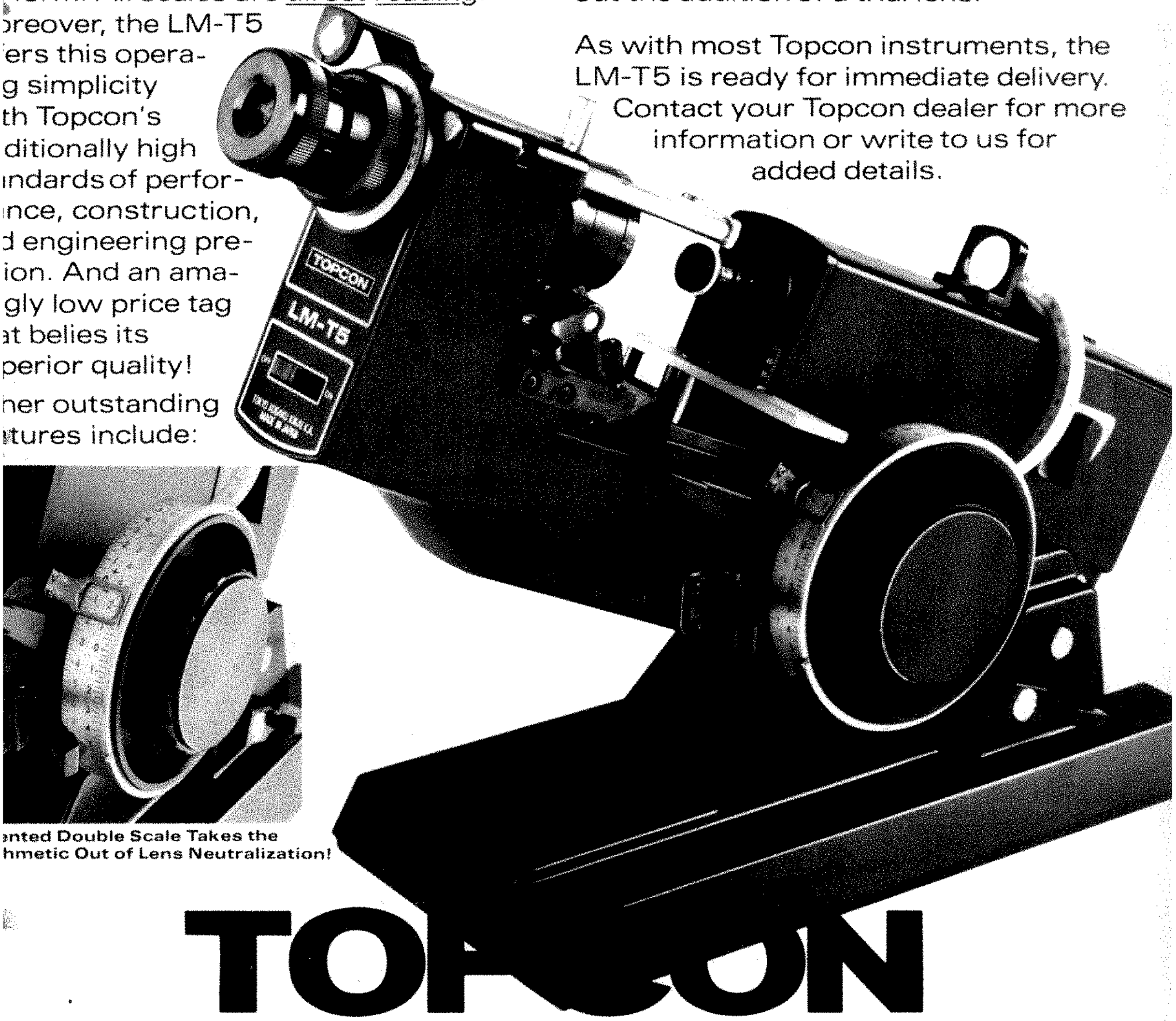

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Interested persons should send curriculum vitae, or contact Karen M. Wright, Deputy Personnel Officer, National Eye Institute, National Institutes of Health, Public Health Service, Bethesda, Maryland 20014. Phone (301)496-4274 for further information. Equal Opportunity Employer.

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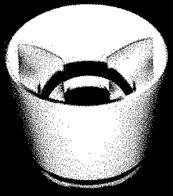
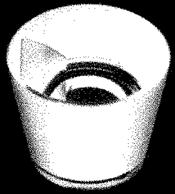
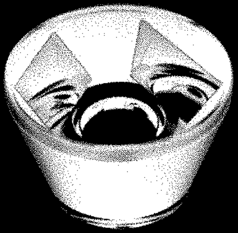


One-mirror gonio lens

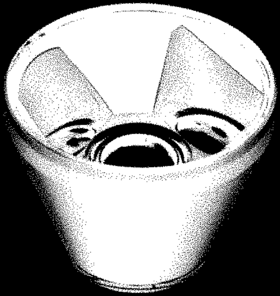


Two-mirror gonio lens

Macula

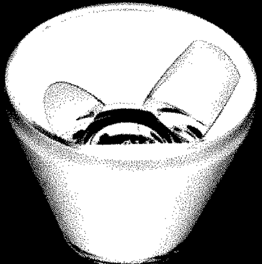


Small Three-mirror lens



Large Three-mirror lens

Three-mirror lens
for babies



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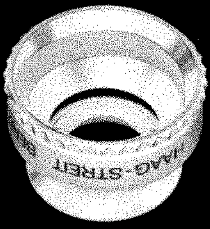
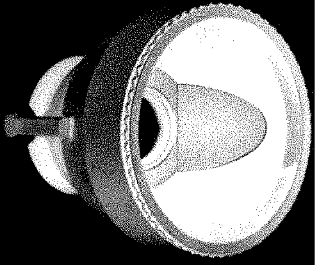


Laser Macula lens



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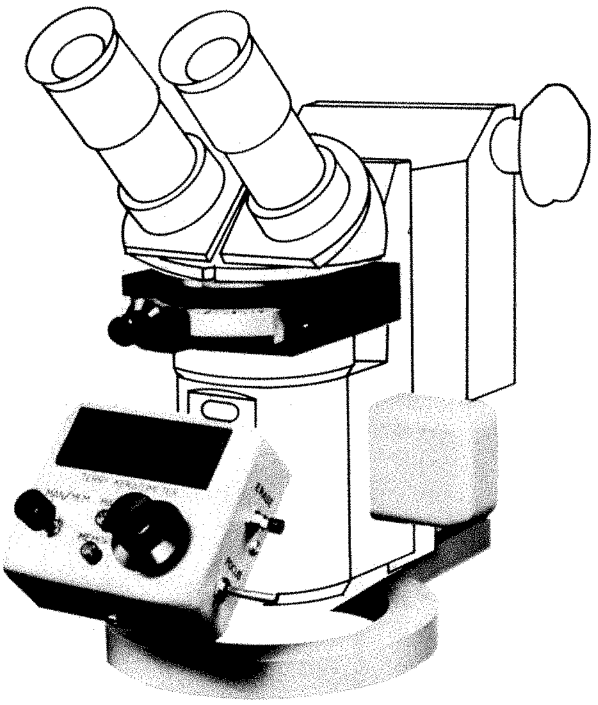

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Three base curves, one diameter provide ample range to fit a majority of patients. NATURVUE'S® unique flexible flange and just three fundamental base curves in one diameter, 14.5mm, will provide sufficient sagittal variance (0.48mm) to fit most of your patients.

Unique flexible flange maintains centering and helps tear exchange.

NATURVUE'S® beveled flange gently hugs the conjunctiva, maintaining centration and promoting tear exchange. It conforms to individual corneal and scleral configurations and allows a majority of patients to be fitted with one of three basic NATURVUE® Lenses.

LASERTRACE® marks help evaluate movement accurately and quickly, greatly simplifying precise fitting.

Each NATURVUE® lens is inscribed with marks near the periphery. They offer perfect identification, more precise, easier fitting. Focus on the LASERTRACE marks to evaluate lens movement and relate them to a fixed target such as a conjunctival vessel.

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Patents pending—NATURVUE® Lens with laser marks

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Milton Roy Company, Ophthalmic Group • P.O. Box 1899, Sarasota, FL 33578

NATURVUE® with LASERTRACE®
(hefilcon A) Soft Lens

NATURVUE® (hefilcon A) CONTACT LENS fabricated from PHP* polymer (poly[2-hydroxyethyl methacrylate] 55%, Water 45%).

DESCRIPTION—The NATURVUE® (hefilcon A) Contact Lens PHP* is a hemispherical shell of approximately 14 mm chord diameter and 0.1 to 1.0 mm thickness. The lens material, hefilcon A, is a hydrophilic random copolymer of 2-hydroxyethyl methacrylate and N-vinyl-2-pyrrolidone. The polymer is a 3 dimensional network of copolymer chains joined by ethylene glycol dimethacrylate crosslinks at a density of about one crosslink for every 70 monomer units. In consists of 55% hefilcon A and 45% water by weight when immersed in normal saline.

ACTIONS—In its hydrated state a NATURVUE® (hefilcon A) Contact Lens PHP* becomes soft and pliable. When placed on the human cornea the hydrated lens acts as a refracting medium to compensate spherical ametropias. The material has a refractive index of 1.43.

INDICATIONS—NATURVUE® (hefilcon A) Contact Lens PHP* are indicated for the correction of visual acuity in persons with nondiseased eyes who have spherical ametropias, refractive astigmatism of 1.50 diopters or less and/or corneal astigmatism of 2.00 diopters or less.

CONTRAINDICATIONS—NATURVUE® (hefilcon A) Contact Lens PHP* are contraindicated by the presence of any of the following conditions:

1. Acute and subacute inflammations of the anterior segment of the eye.
2. Any eye disease which affects the cornea or conjunctiva.
3. Insufficiency of lacrimal secretion.
4. Corneal hypoesthesia.
5. Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Medicants and Eye Drops: The NATURVUE® (hefilcon A) Contact Lens PHP* must be stored ONLY in BOILnSOAK® or FLEXSOL® solution, depending on the system for disinfection used. No ophthalmic solutions or medicants, including conventional hard contact lens solutions, can be used by NATURVUE® (hefilcon A) Contact Lens PHP* wearers prior to, or while, the lens is in place on the eye. Only the recommended disinfecting soaking solutions BOILnSOAK® or FLEXSOL® the recommended cleaner, PREFLEX® or the recommended lubricating solution ADAPETTES® may be used on the NATURVUE® (hefilcon A) Contact Lens PHP.*

Abrasions and Infections: If the lenses become less comfortable to the wearer than when they were first placed on the wearer's corneas, this may indicate the presence of a foreign body. The lenses should be removed immediately and the patient examined. If any scratching, redness or pain is present, a physician should be consulted immediately.

Lens Sanitation: Patients must adhere to recommended daily sanitary care of the NATURVUE® (hefilcon A) Contact Lens PHP.* Failure to follow this procedure may result in development of serious ocular infections.

Wearing Restrictions: The NATURVUE® (hefilcon A) Contact Lens PHP* should be removed before sleeping or swimming or in the presence of noxious and irritating vapors.

Visual Blurring: When visual blurring occurs the lens must be removed until the condition subsides.

PRECAUTIONS

Storage: NATURVUE® (hefilcon A) Contact Lens PHP* must be stored ONLY in BOILnSOAK® or FLEXSOL® solution. If left exposed to air, the lenses will dehydrate. If a lens dehydrates, it should be soaked in BOILnSOAK® or FLEXSOL® solution until it returns to a soft, supple state.

CLEANING AND DISINFECTING: NATURVUE® (hefilcon A) Contact Lens PHP* must be BOTH cleaned and disinfected daily. One procedure does not replace the other. CLEANING is necessary to remove mucus and film from the lens surface. This can be accomplished by using PREFLEX® Cleaning Solution on a daily basis. Excessive deposits may damage the lens, therefore, if this occurs, review care of lens with patient.

Thermal Disinfection: Disinfection with the NATURVUE® Thermal Unit and BOILnSOAK® solution has been shown to be an effective disinfection system for the daily care of the NATURVUE® (hefilcon A) Contact Lens PHP.* NATURVUE® (hefilcon A) Contact Lens PHP* must be cleaned daily with PREFLEX® Cleaning Solution. Fresh BOILnSOAK® solution must be used daily for storing the lenses. The NATURVUE® Carrying Case must be emptied and refilled with fresh BOILnSOAK® solution just before disinfecting the lenses.

If a NATURVUE® Thermal Unit is temporarily not available for disinfecting the lenses, the lenses may be boiled in the carrying case in a pan of water for 10 minutes.

Chemical Disinfection: Disinfection with PREFLEX® FLEXSOL® and NORMOL® has also been shown to be an effective disinfection system for the daily care of the NATURVUE® (hefilcon A) Contact Lenses PHP.* NATURVUE® (hefilcon A) Contact Lenses PHP* must be cleaned and rinsed daily (or after wearing) with PREFLEX® and NORMOL® The storage case must be emptied and refilled with fresh FLEXSOL® when disinfecting the lenses. Fresh FLEXSOL® must be used daily for storing and disinfecting the lenses.

WARNING: DO NOT MIX OR ALTERNATE THE DISINFECTION AND STORAGE SYSTEMS. FLEXSOL® SHOULD NOT BE USED WITH HEAT.

Hygiene—Patients must be instructed to wash and rinse hands thoroughly and dry with a lint-free towel before handling the lenses. Cosmetics, lotions, soaps and creams must not come in contact with the lenses because eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until hair spray has settled.

Fluorescein—Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with normal saline solution and wait at least one hour before replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescein irreversibly.

ADVERSE REACTIONS—Serious corneal damage may result from wearing a lens which has been soaked in a conventional hard contact lens solution.

Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritations.

On rare occasions a lens may adhere to an eye as a result of the patient sleeping with the lens on or wearing a hypotonic lens. If a lens adheres for any reason, patients may be instructed to apply a few drops of BOILnSOAK® or ADAPETTES® solution and wait until the lens moves freely before removing it.

Rainbows or halos around objects or blurring of the vision may occur if the lenses are worn continuously for too long a time. Removal of the lenses and a rest period of at least one hour generally relieves these symptoms. Excessive tearing, unusual eye secretions, and photophobia are not normal; if these occur, the patient should be examined by a practitioner to determine their cause.

DOSAGE AND ADMINISTRATION—Conventional methods of fitting hard contact lenses do not apply to NATURVUE® (hefilcon A) Contact Lenses PHP.* For a detailed description of the fitting technique, refer to the NATURVUE® Fitting Guide, additional copies of which are available from:
Milton Roy Company
P.O. Box 1899, Sarasota, Florida 33578.

There may be a tendency for the patient to overwear the lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to the patient:

Day	Maximum Wearing Time (Continuous Hours)	Day	Maximum Wearing Time (Continuous Hours)
1	3	8	8
2	3	9	8
3	4	10	10
4	4	11	12
5	6	12	14
6	6	13	15
7	8	14	all waking hours*

*LENSES SHOULD NEVER BE WORN 24 HOURS A DAY.

Care must be taken on the initial visit to assure that the patient is supplied with a NATURVUE® Patient Care Kit and fully understands all care and handling instructions for the lenses. As with any contact lens, regular recall visits are necessary to assure patient health and compliance with instructions.

In Use Durability: With normal use and patient care NATURVUE® (hefilcon A) Contact Lenses PHP* have been shown to be durable for a minimum of one year.

HOW SUPPLIED: Each lens is supplied sterile in a glass vial containing sterile normal saline solution. The glass vial is marked with the base curve, dioptric power, diameter, thickness and the manufacturing lot number of the lens.

To assure proper lens care and handling, each NATURVUE® patient must be supplied with a complete NATURVUE® Patient Care Kit.

The NATURVUE® Patient Care Kit is a package required for lens cleaning, disinfecting and storing consisting of the following:

Thermal Disinfection
System Package contains:
NATURVUE® Carrying
and Storage Case
NATURVUE® Thermal Unit
Soaking Solution:
Cleaning Solution
Patient Instruction Manual
Package Insert

Chemical Disinfection
System Package contains:
NATURVUE® Storage Case Cleaning
Solution: PREFLEX®
Rinsing Solution: NORMOL®
Storage and Disinfection Solution:
FLEXSOL®
Lubricating Solution: ADAPETTES®
Patient Instruction Manual
Package Insert

MILTON ROY

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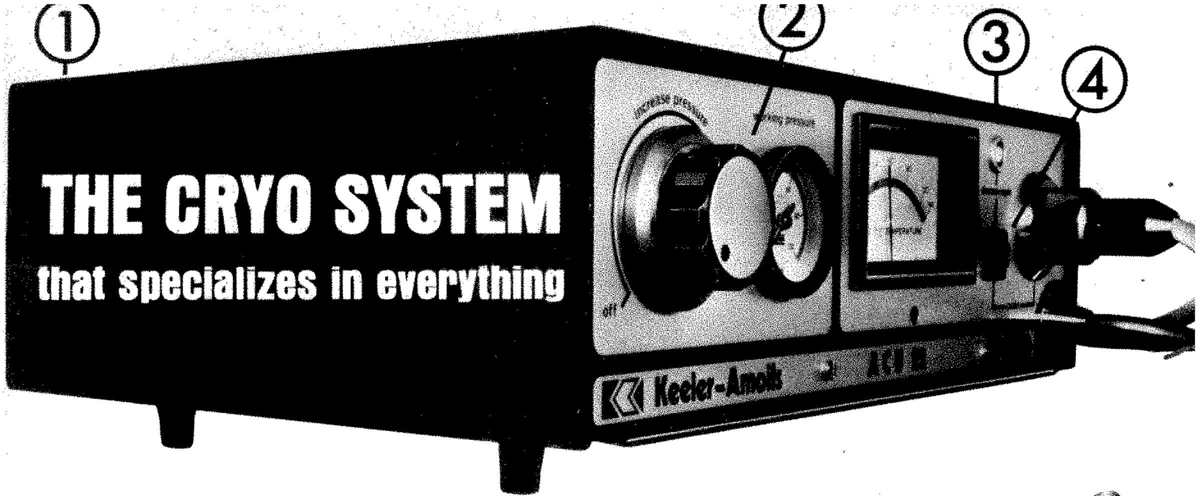

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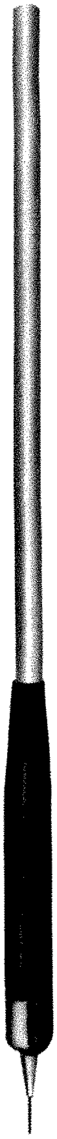
that specializes in everything



For the ophthalmologist (and everyone else), Keeler introduces a cryo system that makes the rounds . . . the new ACU22 cryosurgery unit.

Non-electric for instant freeze and defrost, the new ACU22 provides a variety of important, innovative features. And, using specially-designed Keeler/Amoils probes, the ACU22 performs as a multi-purpose cryosurgery/cryotherapy system, with applications for a broad range of medical specialties.

All ACU22 ophthalmic probes can also be used with the Keeler/Amoils ACU20 non-electric ophthalmic cryo unit (available at a slightly lower cost). But, for a cryosurgery system that's at home in any O.R., call or write for details on the new ACU22.

➊ Exhaust vent on back of console guides coolant gases (CO_2 or N_2O) out of the O.R. (via tubing).

➋ Pressure regulator knob on console makes remote adjustment on gas cylinders unnecessary.

➌ Outlet for external thermocouple, which monitors temperature when inserted in adjacent tissue.

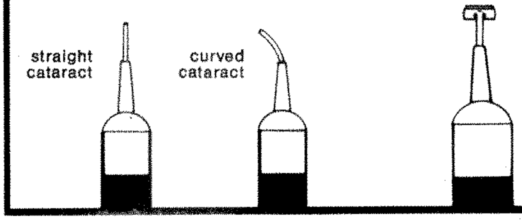
➍ Switch converts probe-tip temperature meter into surrounding-tissue temperature meter.

APPLICATIONS: 19 different probes for use in ophthalmology, ENT, gynecology, proctology, dermatology, oral/dental and general surgical procedures.

NEW - Available for electric and non-electric.

MICRO (1MM) PROBES

MACHEMER/BIETTI PROBE
larger (6mm x 3mm) tip reduces time for multiple application procedures.



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All products repaired and serviced in Broomall, Penna.

Keeler

...with the future in sight

Remember those patients who took forever learning insertion and constantly damaged or lost their lenses?
Our files are jammed with thank you letters from happy patients who have stopped dropping, losing, contaminating and damaging their lenses.

POSITIVE SOFT LENS INSERTION

Soft-Sert™ holds the lens securely, without suction, even upside down...yet it releases the lens automatically on easy contact with the eye. That rubber cup is soft and resilient too, so it's really easy on the eye and lens. During insertion, it provides peripheral support, preventing the soft lens from inverting.

WHY YOUR PATIENTS NEED Soft-Sert™

- Provides safe, aseptic insertion.
- Helps prevent loss and damage.
- Prevents soft lenses from inverting.
- Eliminates long fingernail problems.
- Ideal for all lenses including ultra-thin and post-cataract.

Soft-Sert™ is also a very professional, aseptic means for the practitioner or assistant to insert soft lenses during the fitting procedure.

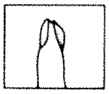
LETTERS. . .WE GET LETTERS

Including some glowing testimonials from some of the more than 10,000 eye care professionals now using Soft-Sert™. We'll send you a sampling of those letters if you'd like. Or if you can't wait, why not order today.

Soft-Sert™

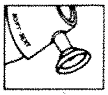
U.S. and Foreign Patents Pending.

No more inversion



How often does this happen to your patients?

SOFT-SERT™ holds the lens securely

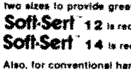


The soft lens is secure (without suction), even when held upside-down

Who Needs SOFT-SERT™?

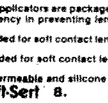
APHAKES HYPEROPES ULTRA-THIN lens patients

Save On Chair Time



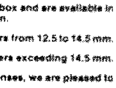
Remember that patient that took two or three visits just to teach insertion

SOFT-SERT™ is easily disinfected



Aseptic insertion

Our novel unifit finger attachment



Allows both hands to manipulate the eyelids, if necessary

How to order Soft-Sert™

Soft-Sert™ soft contact lens applicators are packaged six to a box and are available in two sizes to provide greater efficiency in preventing lens inversion.

Soft-Sert™ **12** is recommended for soft contact lens diameters from 12.5 to 14.5 mm.

Soft-Sert™ **14** is recommended for soft contact lens diameters exceeding 14.5 mm.

Also, for conventional hard, gas permeable and silicone contact lenses, we are pleased to announce the availability of **Soft-Sert**™ **8.**

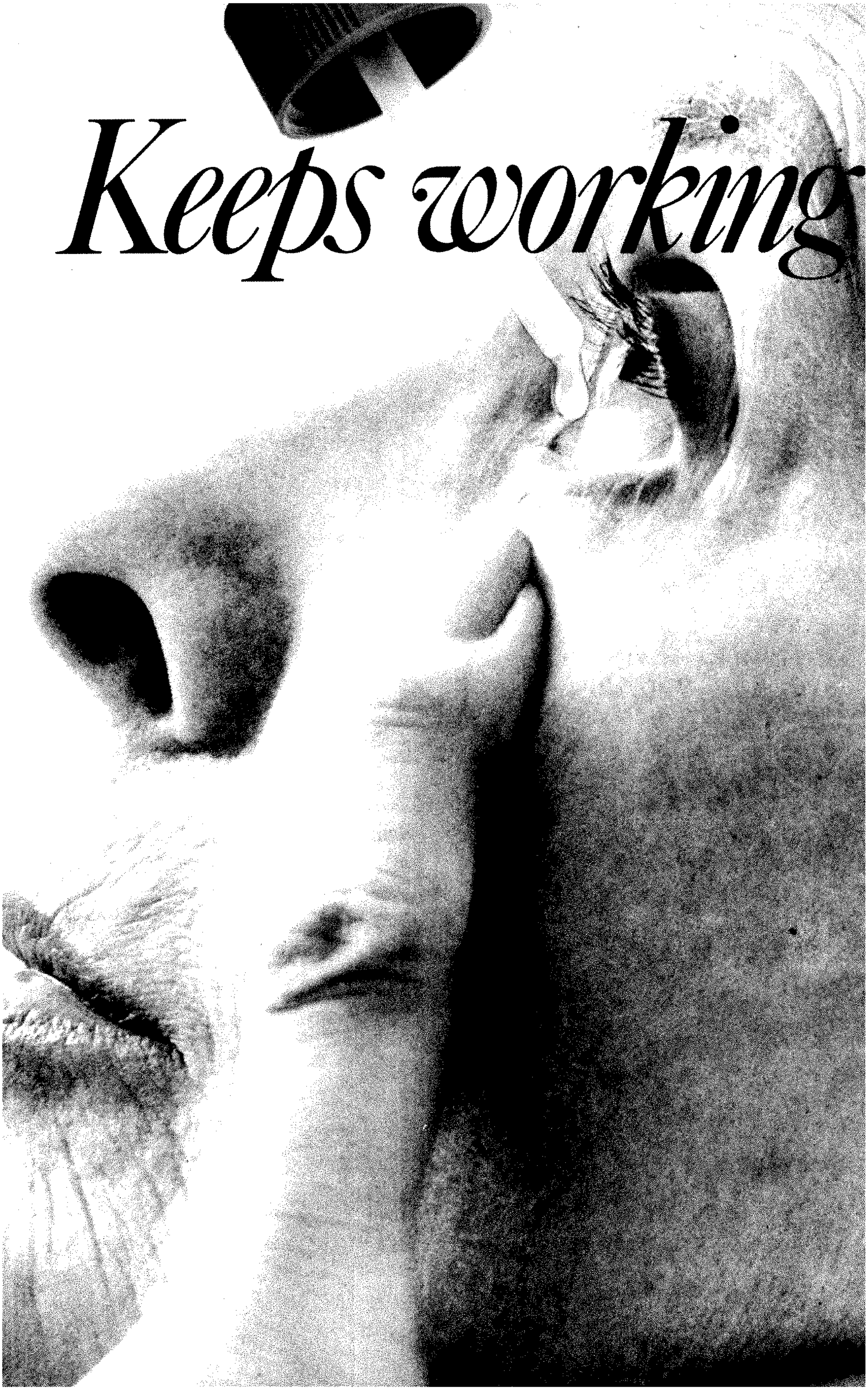
Soft-Sert, INC.
P.O. Box 376
Cedarhurst, N.Y. 11516

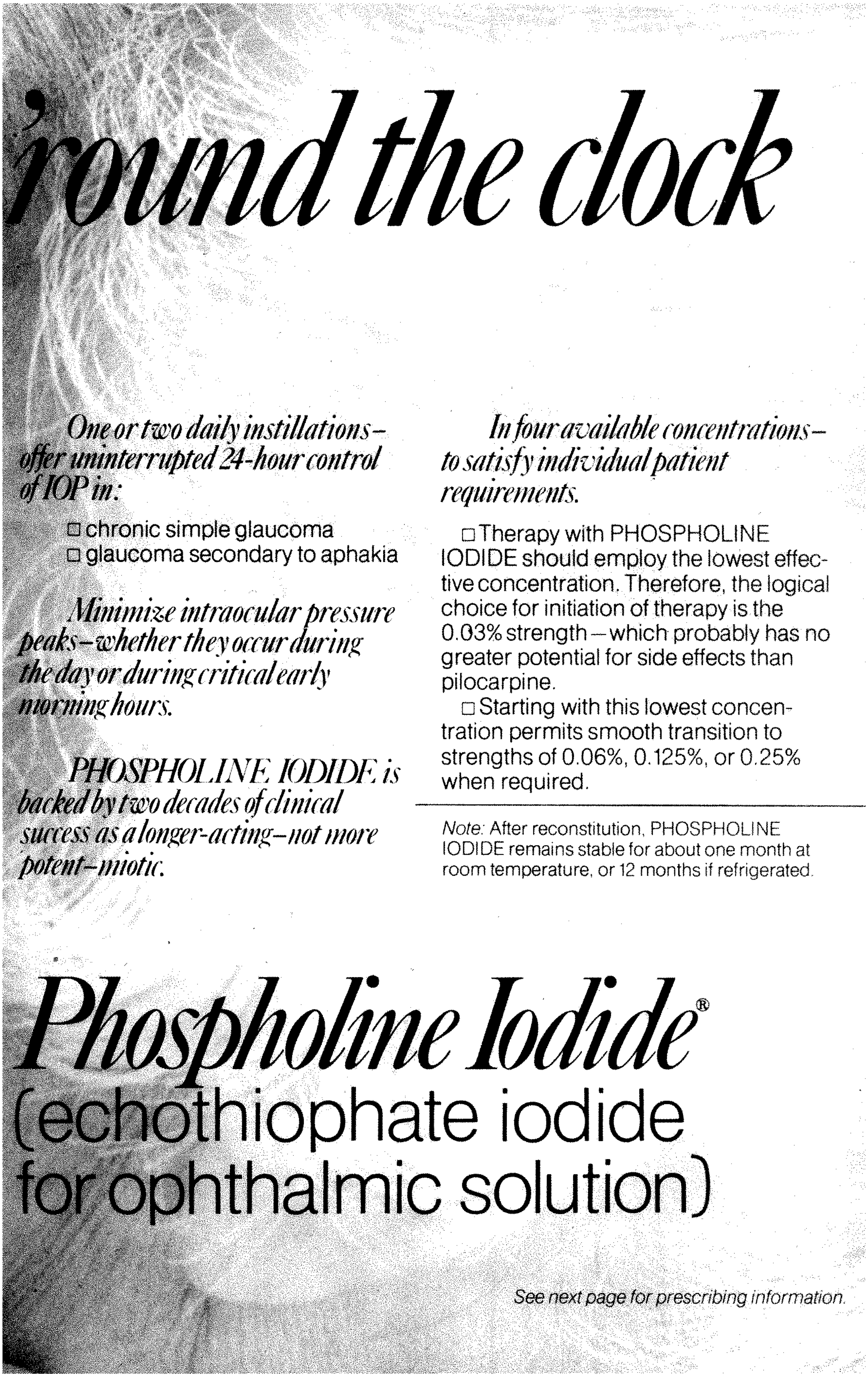
Please Send Me The Following:

_______ Box(es) of SOFT-SERT™ 12 (at $13.50 per box of 6)
_______ Box(es) of SOFT-SERT™ 14 (at $13.50 per box of 6)
_______ Box(es) of SOFT-SERT™ 8 (at $13.50 per box of 6)

NAME ____________________
ADDRESS ____________________
CITY ____________ STATE ________ ZIP ________

Postage and handling included.
Check or Money Order MUST accompany order.


Keeps working



'round the clock

One or two daily instillations–offer uninterrupted 24-hour control of IOP in:

☐ chronic simple glaucoma
☐ glaucoma secondary to aphakia

Minimize intraocular pressure peaks–whether they occur during the day or during critical early morning hours.

PHOSPHOLINE IODIDE is backed by two decades of clinical success as a longer-acting–not more potent–miotic.

In four available concentrations–to satisfy individual patient requirements.

☐ Therapy with PHOSPHOLINE IODIDE should employ the lowest effective concentration. Therefore, the logical choice for initiation of therapy is the 0.03% strength–which probably has no greater potential for side effects than pilocarpine.

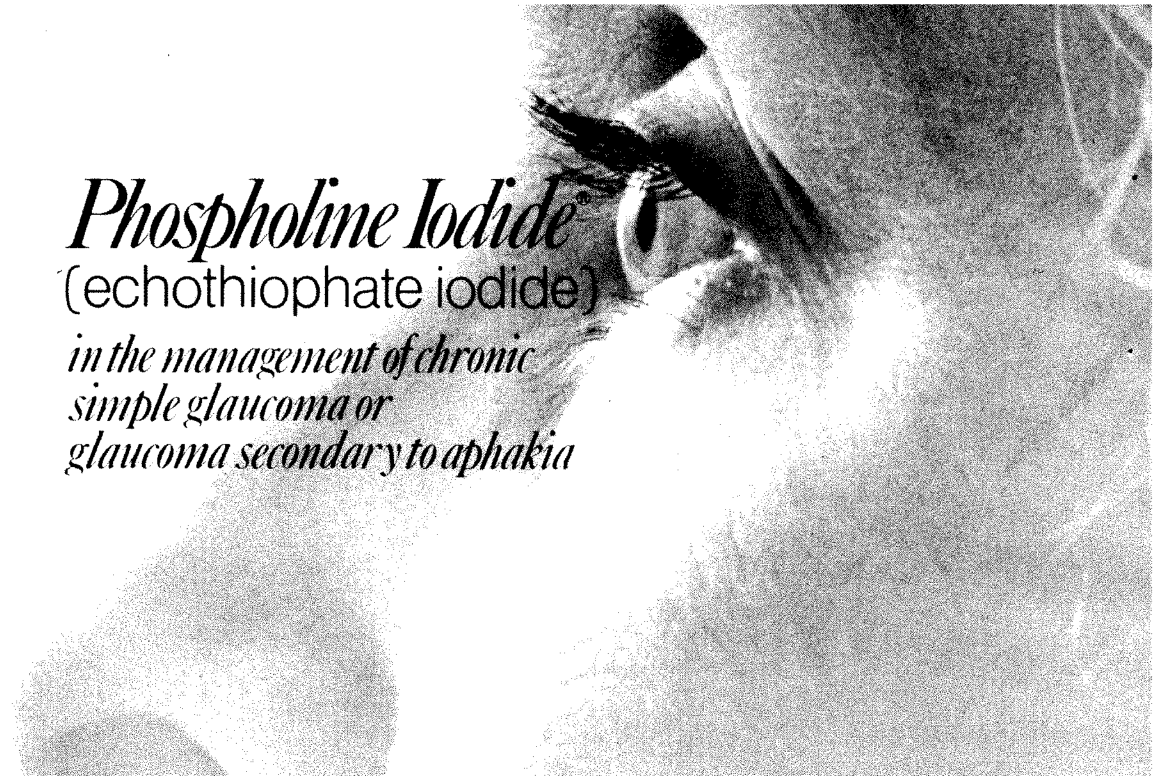
☐ Starting with this lowest concentration permits smooth transition to strengths of 0.06%, 0.125%, or 0.25% when required.

Note: After reconstitution, PHOSPHOLINE IODIDE remains stable for about one month at room temperature, or 12 months if refrigerated.

Phospholine Iodide®

(echothiophate iodide for ophthalmic solution)

See next page for prescribing information.



Phospholine Iodide®
(echothiophate iodide)
in the management of chronic simple glaucoma or glaucoma secondary to aphakia

BRIEF SUMMARY
(For full prescribing information, see package circular.)
PHOSPHOLINE IODIDE*
(ECHOTHIOPHATE IODIDE FOR OPHTHALMIC SOLUTION)

PHOSPHOLINE IODIDE is a long-acting cholinesterase inhibitor for topical use.

Indications: Glaucoma—Chronic open-angle glaucoma. Subacute or chronic angle-closure glaucoma after iridectomy or where surgery is refused or contraindicated. Certain non-uveitic secondary types of glaucoma, especially glaucoma following cataract surgery.

Accommodative esotropia—Concomitant esotropias with a significant accommodative component.

Contraindications: 1. Active uveal inflammation.

2. Most cases of angle-closure glaucoma, due to the possibility of increasing angle block.

3. Hypersensitivity to the active or inactive ingredients.

Warnings: 1. *Use in Pregnancy.* Safe use of anticholinesterase medications during pregnancy has not been established, nor has the absence of adverse effects on the fetus or on the respiration of the neonate.

2. Succinylcholine should be administered only with great caution, if at all, prior to or during general anesthesia to patients receiving anticholinesterase medication because of possible respiratory or cardiovascular collapse.

3. Caution should be observed in treating glaucoma with PHOSPHOLINE IODIDE in patients who are at the same time undergoing treatment with systemic anticholinesterase medications for myasthenia gravis, because of possible adverse additive effects.

Precautions: 1. Gonioscopy is recommended prior to initiation of therapy.

2. Where there is a quiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and ciliary muscle contraction that may occur.

3. While systemic effects are infrequent, proper use of the drug requires digital compression of the nasolacrimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

4. Temporary discontinuance of medication is necessary if salivation, urinary incontinence, diarrhea, profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities occur.

5. Patients receiving PHOSPHOLINE IODIDE who are exposed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin. During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable.

6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer, pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.

7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.

8. PHOSPHOLINE IODIDE (echothiophate iodide) should be used with great caution, if at all, where there is a prior history of retinal detachment.

Adverse Reactions: 1. Although the relationship, if any, of retinal detachment to the administration of PHOSPHOLINE IODIDE has not been established, retinal detachment has been reported in a few cases during the use of PHOSPHOLINE IODIDE in adult patients without a previous history of this disorder.

2. Stinging, burning, lacrimation, lid muscle twitching, conjunctival and ciliary redness, browache, induced myopia with visual blurring may occur.

3. Activation of latent iritis or uveitis may occur.

4. Iris cysts may form, and if treatment is continued, may enlarge and obscure vision. This occurrence is more frequent in children. The cysts usually shrink upon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Rarely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative esotropia.

5. Prolonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.

6. Lens opacities occurring in patients under treatment for glaucoma with PHOSPHOLINE IODIDE have been reported and similar changes have been produced experimentally in normal monkeys. Routine examinations should accompany clinical use of the drug.

7. Paradoxical increase in intraocular pressure may follow anticholinesterase instillation. This may be alleviated by prescribing a sympathomimetic mydriatic such as phenylephrine.

Overdosage: Antidotes are atropine, 2 mg. parenterally; PROTOPAM* CHLORIDE (pralidoxime chloride), 25 mg. per kg intravenously; artificial respiration should be given if necessary.

How Supplied: Four potencies are available. 1.5 mg. package for dispensing 0.03% solution; 3.0 mg. package for 0.06% solution; 6.25 mg. package for 0.125% solution; 12.5 mg. package for 0.25% solution. Also contains potassium acetate (sodium hydroxide or acetic acid may have been incorporated to adjust pH during manufacturing), chlorobutanol (chloral derivative), mannitol, boric acid and exsiccated sodium phosphate.

The Ophthalmos Division
AYERST LABORATORIES
New York, N.Y. 10017

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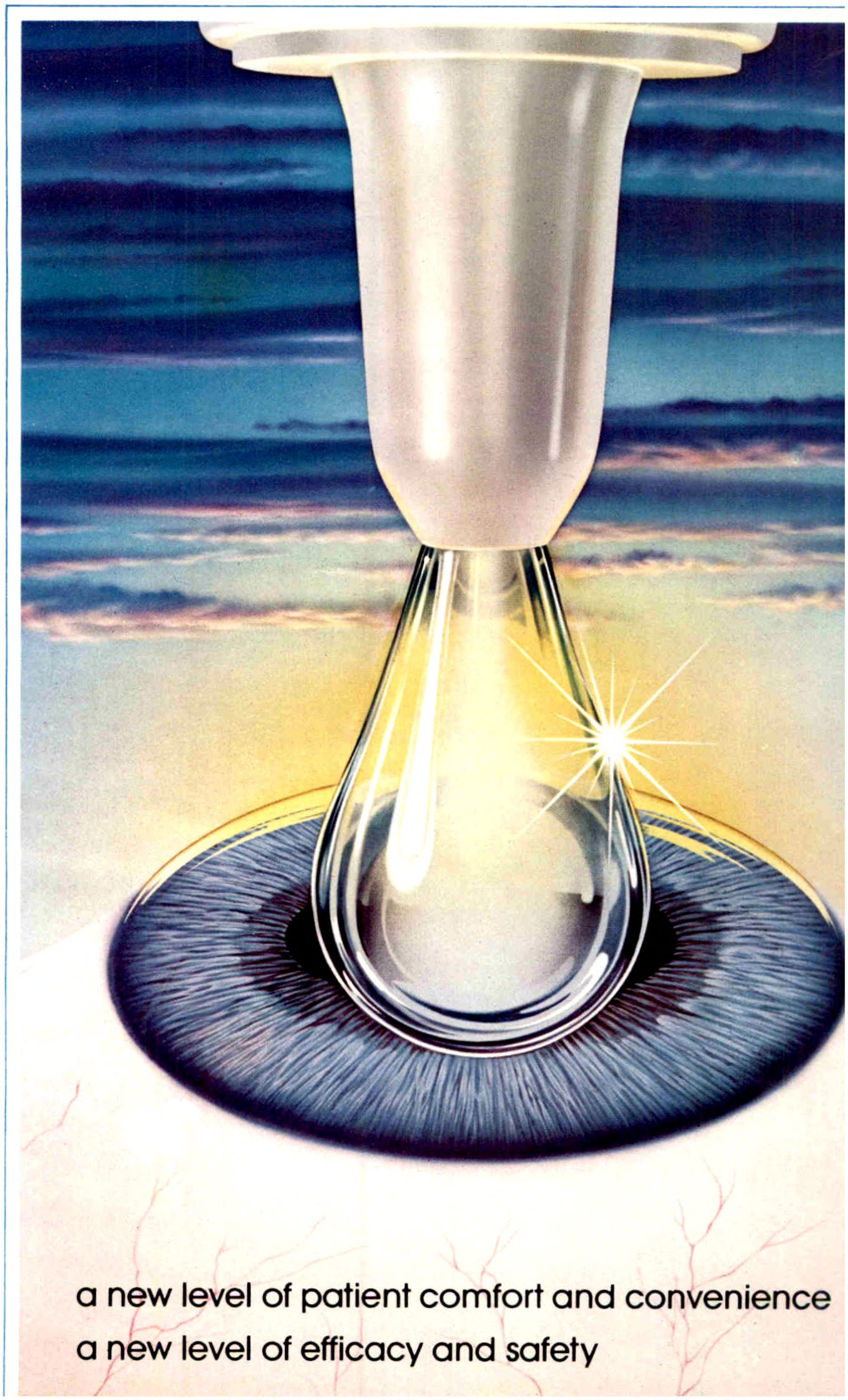
Merck Sharp & Dohme announces

a significant advance in the topical treatment of chronic open-angle glaucoma...


a new level of patient comfort and convenience
a new level of efficacy and safety

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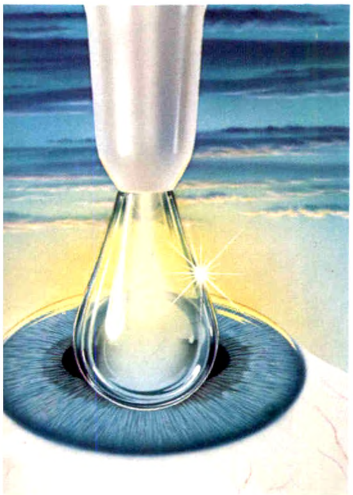
For a brief summary of
Prescribing Information,
please see last page
of this advertisement.



new

Timoptic™

(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION

a beta-adrenergic blocking agent
in ophthalmic solution...for reduction of
elevated IOP in chronic open-angle glaucoma

significantly more effective than pilocarpine or
epinephrine with fewer and less severe side effects*

- little or no change in pupil size
- little or no blurring of vision
- no dimming of vision...no night blindness
- little or no effect on tear production
- little or no hyperemia
- simple "one-drop" b.i.d. dosage (may encourage long-term compliance)

TIMOPTIC™ (Timolol Maleate, MSD) is contraindicated in patients who are hypersensitive to any component of this product.

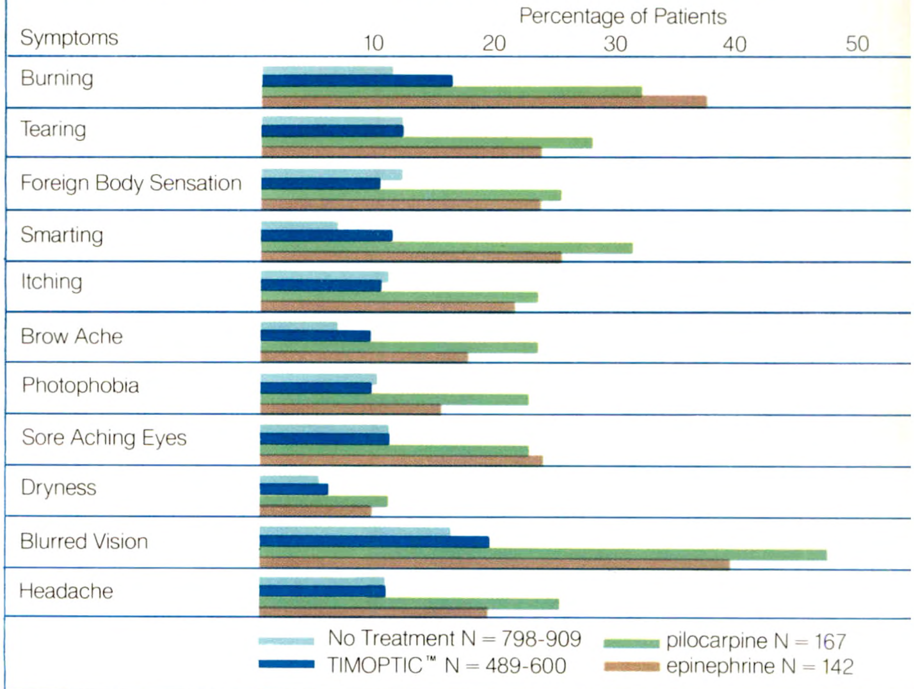
Occasionally, mild ocular irritation has been reported.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.

*Based on controlled multiclinic studies, evaluating over 1,000 patients. Data on file, Merck Sharp & Dohme. For published articles from the clinical studies on TIMOPTIC,™ please see last page of this advertisemen

a new level of patient comfort and convenience

In clinical studies—a much lower incidence of ocular discomfort than with pilocarpine or epinephrine



Adverse effects commonly caused by pilocarpine and epinephrine as reported in the literature—rarely encountered with TIMOPTIC™ (Timolol Maleate, MSD)

Drug-Induced Adverse Effects

pilocarpine	**TIMOPTIC™ (in clinical studies)**	**epinephrine**	**TIMOPTIC™ (in clinical studies)**
extreme miosis	none reported	conjunctival irritation	rare
spasm of accommodation	none reported	hyperemia	rare and mild
shallowing of the anterior chamber	none reported	macular edema in aphakic patients	none reported
night blindness and dimming of vision	none reported	pigment deposits on lid margins and conjunctiva	none reported
ciliary and conjunctival congestion	rare and mild		

With TIMOPTIC™ slight reduction of the resting heart rate has been observed in some patients. Local hypersensitivity reactions have occurred rarely.

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For a brief summary of Prescribing Information, please see last page of this advertisement.

new Timoptic™

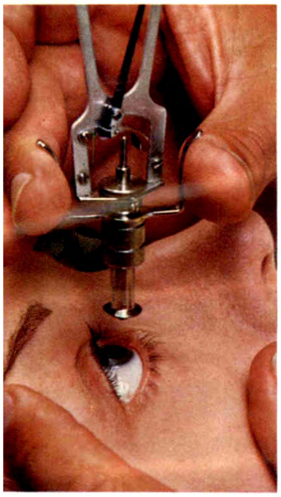
(TIMOLOL MALEATE | MSD) OPHTHALMIC SOLUTION

a beta-adrenergic blocking agent
in ophthalmic solution...for reduction of
elevated IOP in chronic open-angle glaucoma

- Fast onset of action (usually within ½ hour)
- Based on clinical studies of 638 patients maintained on therapy from 14 to 477 days:
  approximately 76% (485 of 638) of patients satisfactorily managed (IOP less than 22 mm Hg) on TIMOPTIC™ alone
  approximately 93% (593 of 638) of patients satisfactorily managed with TIMOPTIC™ alone or administered concomitantly with other antiglaucoma agents
- No evidence of diminished responsiveness reported in 102 patients treated for at least two years
- Absence of miosis may facilitate visualization of optic disc and evaluation of visual fields
- Usually well tolerated
- Produced fewer and less severe side effects than either pilocarpine or epinephrine

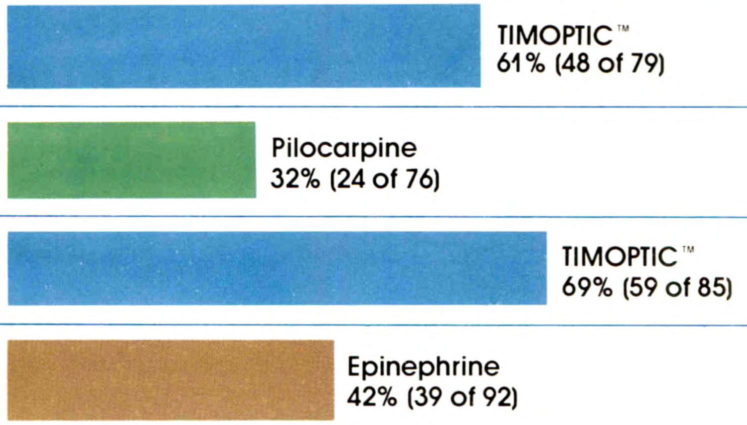
TIMOPTIC™ should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. Patients who are already receiving a beta-adrenergic blocking agent orally should be observed for a potential additive effect.

a new level of efficacy and safety



In comparative clinical studies—significantly more effective than pilocarpine or epinephrine

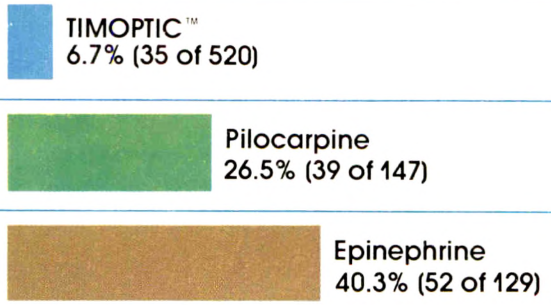
In multiclinic studies, TIMOPTIC™ (Timolol Maleate, MSD) reduced IOP in proportionately more patients



% of patients with IOP reduced to less than 22 mm Hg ($p < 0.01$)

Data are derived from the final visit (week 17) in the multiclinic pilocarpine and epinephrine comparative studies. These include patients who withdrew due to ineffective response or an adverse clinical experience.

In all clinical studies, therapy with TIMOPTIC™ resulted in fewer dropouts* than with pilocarpine or epinephrine



Data include patients participating in clinical studies lasting 14 to 477 days.

*Dropouts are defined as patients whose IOP could not be reduced to levels below 22 mm Hg, or those who withdrew because of an adverse clinical experience.

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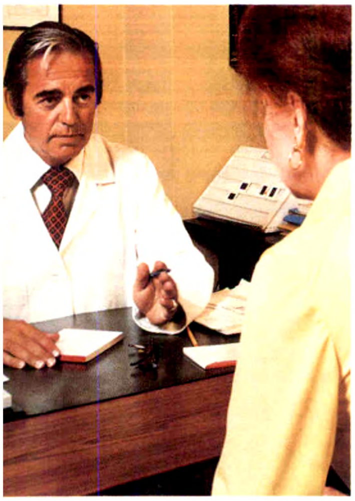
new

Timoptic™

(TIMOLOL MALEATE | MSD) OPHTHALMIC SOLUTION



significantly broadens the medical options in antiglaucoma therapy

- **Permits prolonged maintenance therapy— no evidence of tolerance reported** In a clinical study of 102 patients treated for at least two years, there was no evidence of diminished responsiveness to TIMOPTIC™.
- **Compatible with other agents** Concomitant therapy with pilocarpine and other miotics, and/or epinephrine, and/or systemically administered carbonic anhydrase inhibitors, such as acetazolamide, can be instituted for the patient who needs reduction of IOP greater than provided by one drop of 0.5% TIMOPTIC™ twice a day. Dosages above one drop of 0.5% TIMOPTIC™ twice a day generally have not been shown to produce further reduction in intraocular pressure.
- **Helpful in the medical management of a broader range of patients** In some patients who respond inadequately to maximum available drug therapy, the addition of TIMOPTIC™ may result in satisfactory management of intraocular pressure, thus possibly postponing the need for surgery.
- **Often effective in patients previously difficult to manage**...such as working patients incapacitated by accommodative spasm produced by miotics, older patients with cataracts whose vision is impaired with miotics, patients with heavily pigmented irides whose response to pilocarpine is diminished, and aphakic patients who may develop macular edema from epinephrine.

Since clinical studies in children have not been done, TIMOPTIC™ is not recommended for use in children at this time. TIMOPTIC™ has not been studied in human pregnancy. The use of TIMOPTIC™ in pregnant women requires that the anticipated benefits be weighed against the possible hazards.

a beta-adrenergic blocking agent in ophthalmic solution...for reduction of elevated IOP in chronic open-angle glaucoma

Dosage Guidelines:

How to transfer from current therapy to TIMOPTIC™ (Timolol Maleate, MSD):

1 On the first day: continue with the agent(s) already being used and add one drop 0.25% TIMOPTIC™ in each eye b.i.d.

2 On the second day: discontinue the previously used agent(s) and continue with TIMOPTIC™ in each eye b.i.d.

How to add TIMOPTIC™ to other agents:

1 Administer one drop 0.25% TIMOPTIC™ in each eye b.i.d....while continuing with the agent(s) already being used.

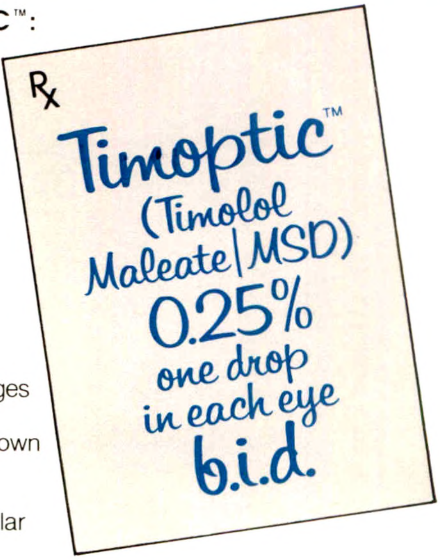
2 If a higher dosage of TIMOPTIC™ is required, substitute one drop 0.5% TIMOPTIC™ in each eye b.i.d....while continuing with the agent(s) already being used.

How to start patients on TIMOPTIC™:

Usual starting dosage:
one drop 0.25% TIMOPTIC™
in each eye twice a day.

If clinical response is not adequate:

Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC™ twice a day in each eye. If the intraocular pressure is maintained at satisfactory levels, the dosage schedules may be changed to one drop once a day in each eye. Dosages above one drop of 0.5% TIMOPTIC™ twice a day generally have not been shown to produce further reduction in IOP.



Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.

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For a brief summary of Prescribing Information, please see following page.

for many patients—a significant advance for the reduction of elevated IOP

- in chronic open-angle glaucoma
- in ocular hypertension
- in aphakic glaucoma
- in some patients with secondary glaucoma

new Timoptic™

(TIMOLOL MALEATE | MSD) OPHTHALMIC SOLUTION

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CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.

PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchial asthma; sinus bradycardia and greater than first-degree block; cardiogenic shock; right ventricular failure secondary to pulmonary hypertension; congestive heart failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockad

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.

Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.

Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.

ADVERSE REACTIONS: Occasionally, signs and symptoms of mild ocular irritation have been reported. Slight reduction of the resting heart rate (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed in some patients. Local hypersensitivity reactions have occurred rarely.

Note: TIMOPTIC has been used in patients with glaucoma wearing conventional (PMMA) hard contact lenses, and has generally been well tolerated. TIMOPTIC has not been studied in patients wearing lenses made with materials other than PMMA.

HOW SUPPLIED:

TIMOPTIC Ophthalmic Solution, 0.25%
TIMOPTIC Ophthalmic Solution, 0.5%

Both are available in 5-ml plastic OCUMETER® Ophthalmic Dispenser with a controlled drop tip.

J8TMOI (DC704600

For more detailed information, consult your MSD Representative or see full Prescribing Informatic

Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, Pa. 19486.

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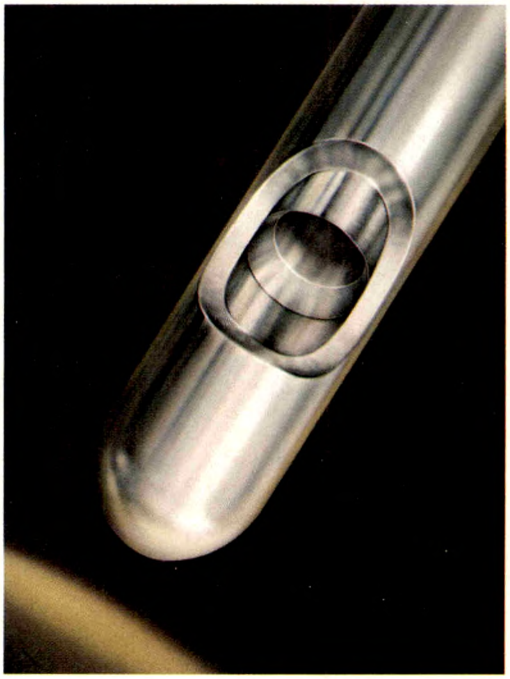
More than a cut above the rest.



The Ocutome®/Fragmatome™ System: Easy to operate. Effortless to maintain. Can be ready for use in less than two minutes and is there when you need it. The only complete system for Anterior Segment Surgery.

Choose from three ways to infuse. Use the *Charles Anterior Segment Infusion Sleeve* when you already have one wound—combines cutting, aspiration, and infusion in one probe. Clean up escaped vitreous quickly.

You will have bimanual control in the anterior chamber with the *Anterior Segment Infusion Pic*—helpful in delaminating lens material and stripping anterior membranes.

Sutured to the globe, the *Infusion Cannulas* will help you maintain IOP with minimal fluid exchange while leaving your hands free. Available in three lengths: 1½ mm (corneal), 2½ mm (standard pars plana), and 4 mm (for aphakic eyes).

Additional advantages. All the best known features are combined in the Ocutome/Fragmatome System. No other unit offers as much. *Guillotine cutting action.* Adjustable cutting port. Variable cutting speed. *Ultrasonic lens fragmenter.* Plus a full line of interchangeable accessory Pics and Probes. *All 20-gauge.* All flash autoclavable. Physicians report that it is the *only truly microsurgical size instrument.*

And there's more. *Completely modular.* You can order what you need now, add on later. *Automatic suction.* Pressure controlled, it gives you suction you can depend on at the exact level you select. And *one simple footswitch* operates it all.

An asset in any hospital. An aid in dealing with cases such as secondary cataract, retro-IOL membrane, inadequate pupil, residual lens material, vitreous loss, capsulectomy, iridectomy, extracapsular cataract removal, pupillary membranectomy, and trauma reconstruction. You are in complete control always. Offer your patients the best there is.

A commitment to quality. Backed by Berkeley's commitment to education through a nationwide program of workshops and the highly acclaimed *Ocutome/Fragmatome Newsletter.* And supported by Berkeley's extensive Research and Development Department, at work today to bring you products that will fill tomorrow's needs.

Learn more about the Ocutome/Fragmatome microsurgery system.

Just check the coupon below, attach to your letterhead, and mail today.

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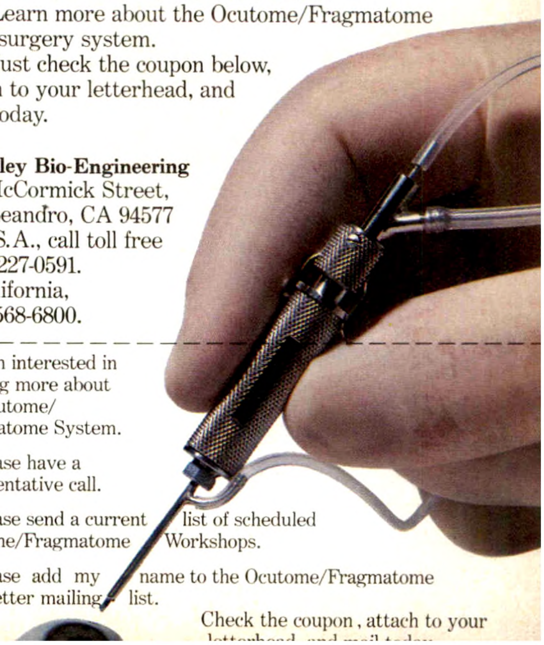

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- Air cooling eliminates the need for expensive plumbing modifications and dependence upon a source of constant water pressure.
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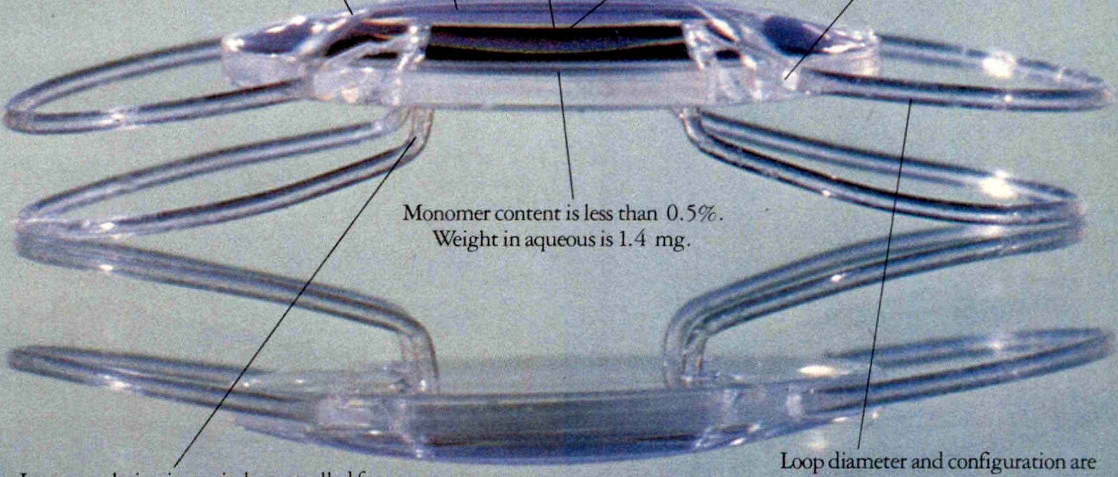

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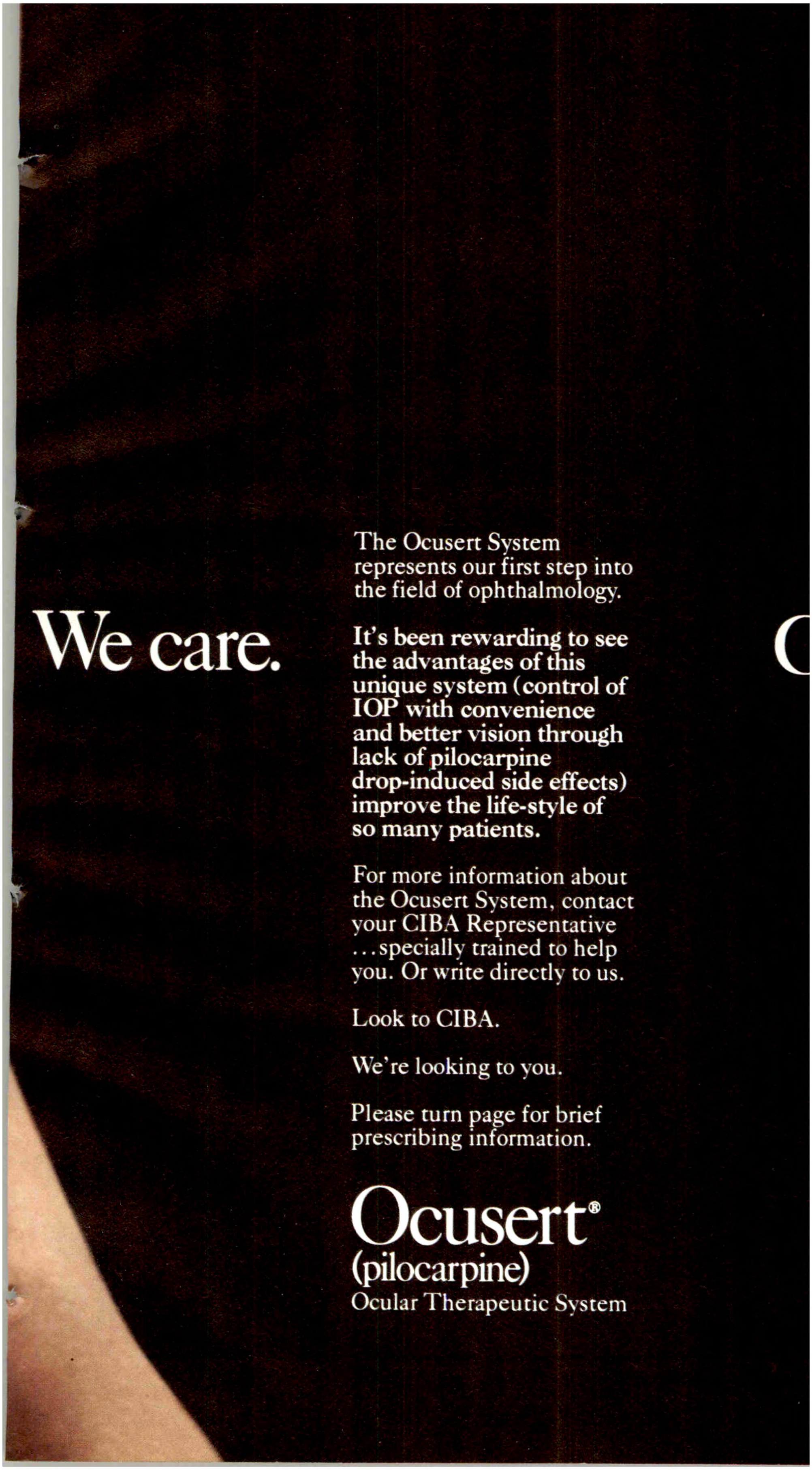

We care.

The Ocusert System represents our first step into the field of ophthalmology.

It's been rewarding to see the advantages of this unique system (control of IOP with convenience and better vision through lack of pilocarpine drop-induced side effects) improve the life-style of so many patients.

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Look to CIBA.

We're looking to you.

Please turn page for brief prescribing information.

Ocusert®
(pilocarpine)
Ocular Therapeutic System

Eye care. You care. We care.

Ocusert® CIBA

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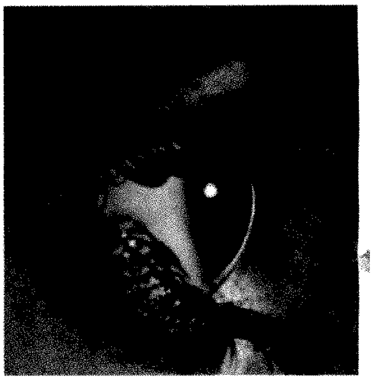

Ocusert® Pilo-20 (pilocarpine)
Ocular Therapeutic System
20 μg/hr. for one week and
Ocusert® Pilo-40 (pilocarpine)
Ocular Therapeutic System
40 μg/hr. for one week

ALZA Product Information

Indications and usage
OCUSERT® pilocarpine system is indicated for control of elevated intraocular pressure in pilocarpine responsive patients. Clinical studies have demonstrated OCUSERT® system efficacy in certain glaucomatous patients.

The patient should be instructed on the use of the OCUSERT® system and should read the package insert instructions for use. The patient should demonstrate to the ophthalmologist his ability to place, adjust and remove the units.

Concurrent Therapy: OCUSERT® systems have been used concomitantly with various ophthalmic medications. The release rate of pilocarpine from the OCUSERT® system is not influenced by carbonic anhydrase inhibitors, epinephrine ophthalmic solutions, fluorescein, or anesthetic, antibiotic or anti-inflammatory steroid ophthalmic solutions. Systemic reactions consistent with an increased rate of absorption from the eye of an autonomic drug, such as epinephrine, have been observed. The occurrence of mild bulbar conjunctival edema which is frequently present with epinephrine ophthalmic solutions is not influenced by the OCUSERT® pilocarpine system.

Contraindications
OCUSERT® pilocarpine system is contraindicated where pupillary constriction is undesirable, such as for glaucomas associated with acute inflammatory disease of the anterior segment of the eye, and glaucomas occurring or persisting after extracapsular cataract extraction where posterior synechiae may occur.

Warnings
Patients with acute infectious conjunctivitis or keratitis should be given special consideration and evaluation prior to the use of the OCUSERT® pilocarpine system.

Damaged or deformed systems should not be placed or retained in the eye. Systems believed to be associated with an unexpected increase in drug action should be removed and replaced with a new system.

Precautions
OCUSERT® pilocarpine system safety in retinal detachment patients and in patients with filtration blebs has not been established. Although ophthalmic solutions have been used effectively in conjunction with the OCUSERT® system, systemic reactions consistent with an increased rate of absorption from the eye of an autonomic drug, such as epinephrine, have been observed. In rare instances, reactions of this type can be severe. The conjunctival erythema and edema associated with epinephrine ophthalmic solutions are not substantially altered by concomitant OCUSERT® pilocarpine system therapy. The use of pilocarpine drops should be considered when intense miosis is desired in certain ocular conditions.

Adverse reactions
Ciliary spasm is encountered with pilocarpine usage but is not a contraindication to continued therapy unless the induced myopia is debilitating to the patient. Irritation from pilocarpine has been infrequently encountered and may require cessation of therapy depending on the judgement of the physician. True allergic reactions are uncommon but require discontinuation of therapy should they occur. Although withdrawal of the peripheral iris from the anterior chamber angle by miosis may reduce the tendency for narrow angle closure, miotics can occasionally precipitate angle closure by increasing the resistance to aqueous flow from posterior to anterior chamber. Miotic agents may also cause retinal detachment; thus, care should be exercised with all miotic therapy especially in young myopic patients.

Some patients may notice signs of conjunctival irritation, including mild erythema with or without a slight increase in mucous secretion when they first use OCUSERT® pilocarpine systems. These symptoms tend to lessen or disappear after the first week of therapy. In rare instances a sudden increase in pilocarpine effects has been reported during system use.

How supplied
OCUSERT® Pilo-20 or Pilo-40 systems are available in packages containing eight individual sterile systems.

Storage and handling
Store under refrigeration (36°-46°F).

Caution
Federal law prohibits dispensing without prescription.

For complete details, including dosage, please see full Prescribing Information.

Manufactured by ALZA Corporation
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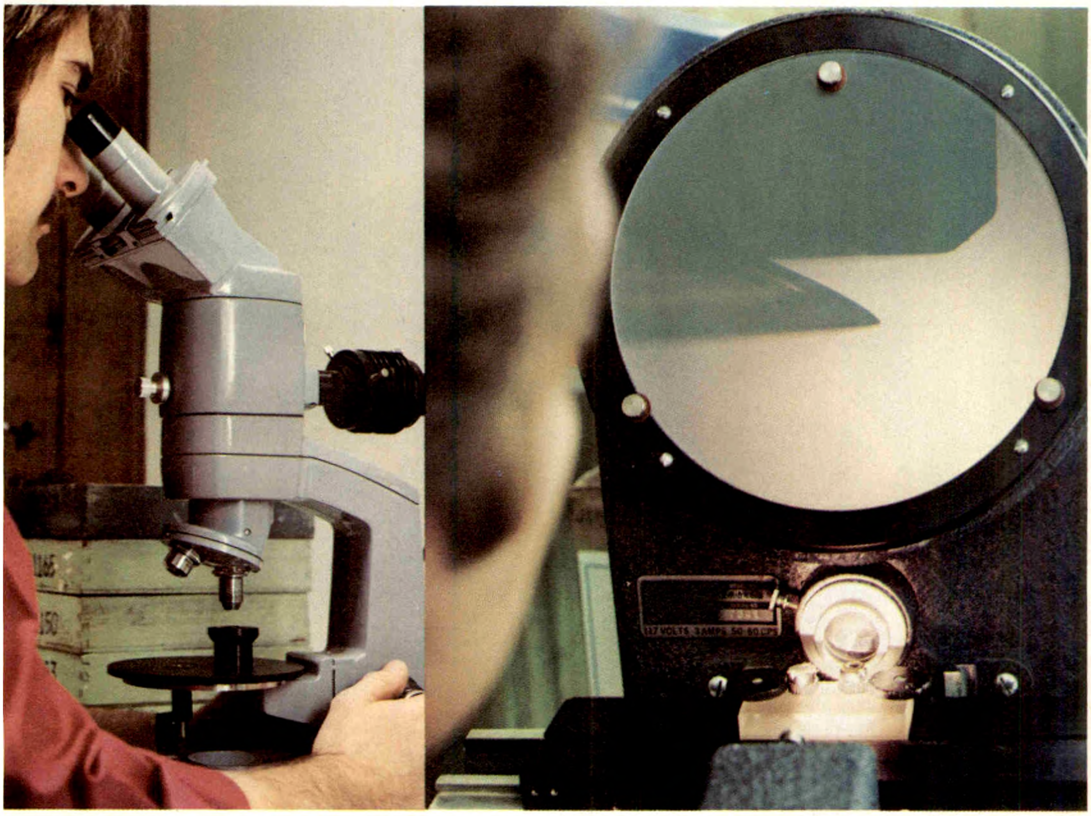

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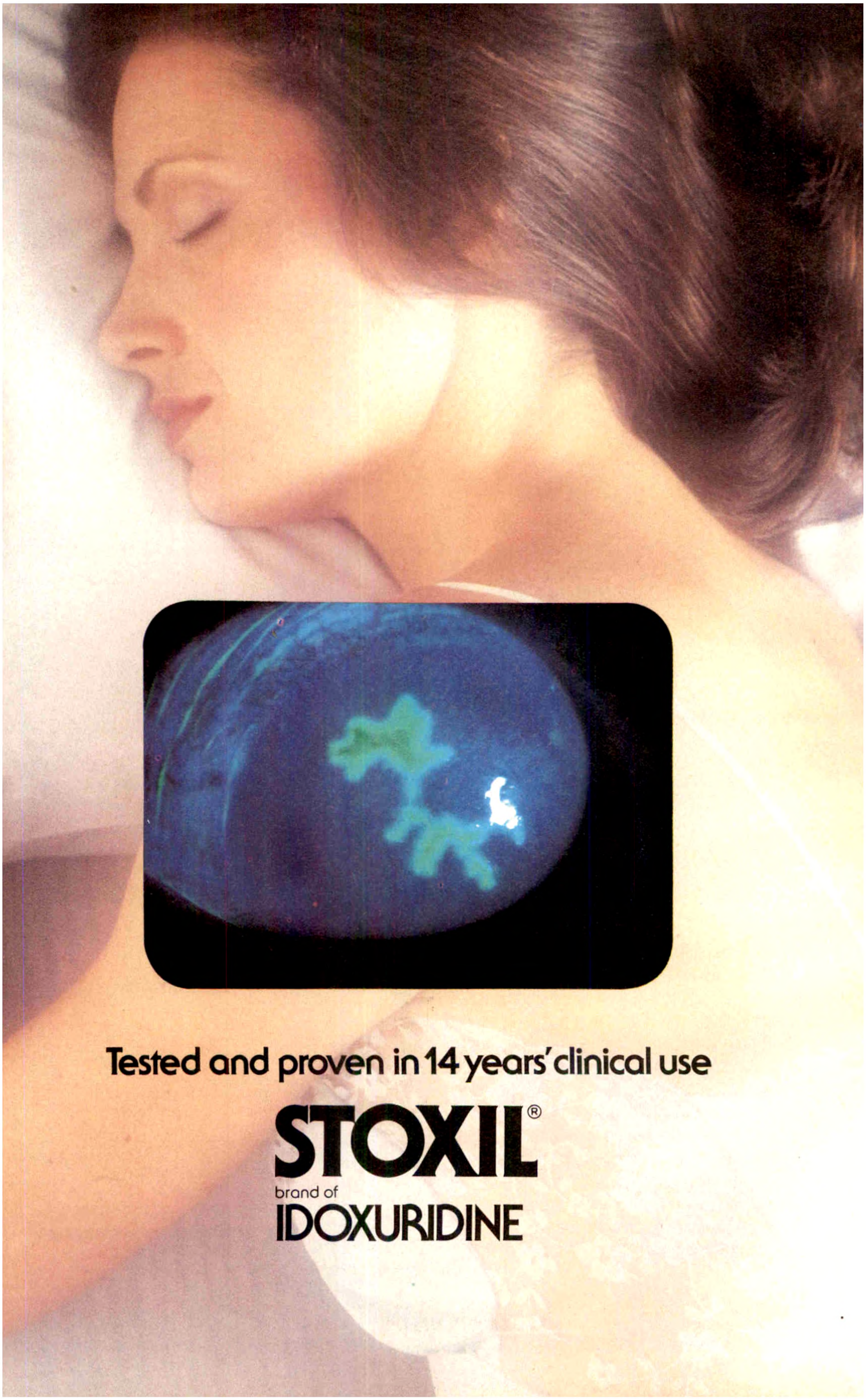
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- Lower Cost than ara-A*

Before prescribing, see complete prescribing information in SK&F literature or PDR. The following is a brief summary.

Indication: Herpes simplex keratitis (topical use only).

Contraindications: Known or suspected hypersensitivity to any of the components.

Warning: Administer with caution in pregnancy or women of childbearing potential. Malformations were reported in one study in rabbits when idoxuridine was instilled in the eyes of the dams; a subsequent, more detailed study in rabbits showed no such effects, even at substantially higher dosages.

Precautions: If there is no response in epithelial infections after 7 or 8 days, other therapy should be considered. Recommended frequency and duration of administration should not be exceeded. Not effective in corneal inflammations if herpes simplex is not present. Boric acid should not be used concomitantly. To insure stability is maintained, the solution should not be mixed with other medications.

Adverse reactions: Occasionally, irritation, pain, pruritus, inflammation, or edema of the eye or lid; rarely, allergic reactions have been reported. Photophobia has occurred. Occasionally, small punctate defects (which may be a manifestation of the infection), corneal clouding, and stippling of the corneal epithelium have been observed.

Supplied: 0.1% Ophthalmic Solution (1 mg./ml.) in 15 ml. bottles with dropper; 0.5% Ophthalmic Ointment (5 mg./gram) in 4 gram tubes.

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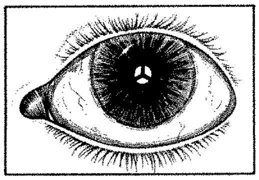
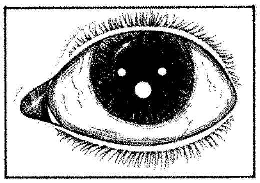
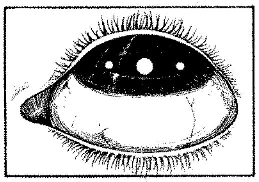


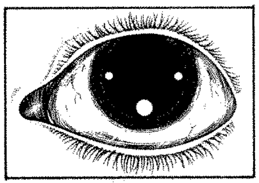
Binocular observation through small or undilated pupils



Routine posterior pole observation through dilated pupils



Optimized view and illumination of fundus periphery



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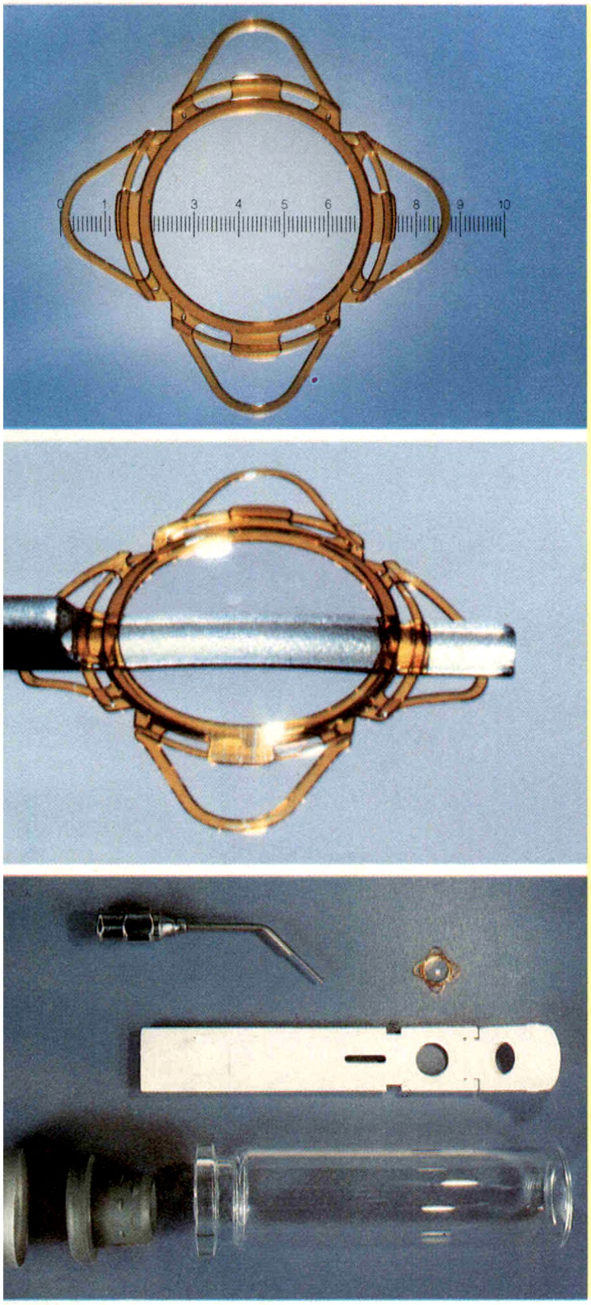

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- each lens packaged with patented insertional instrument
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- autoclavable glass vial
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- precludes need for saline wash

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Detailed information and the forms for obtaining IRC approval and Investigator status are included in our Investigator's Manual, which is available to ophthalmologists on request.

ynell Intraocular Lens* is covered by Patent Nos. 4073014, 4080709, 4118808 and 4122556.
onal domestic and foreign patents and trademarks are pending.

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Dr. ______________________________

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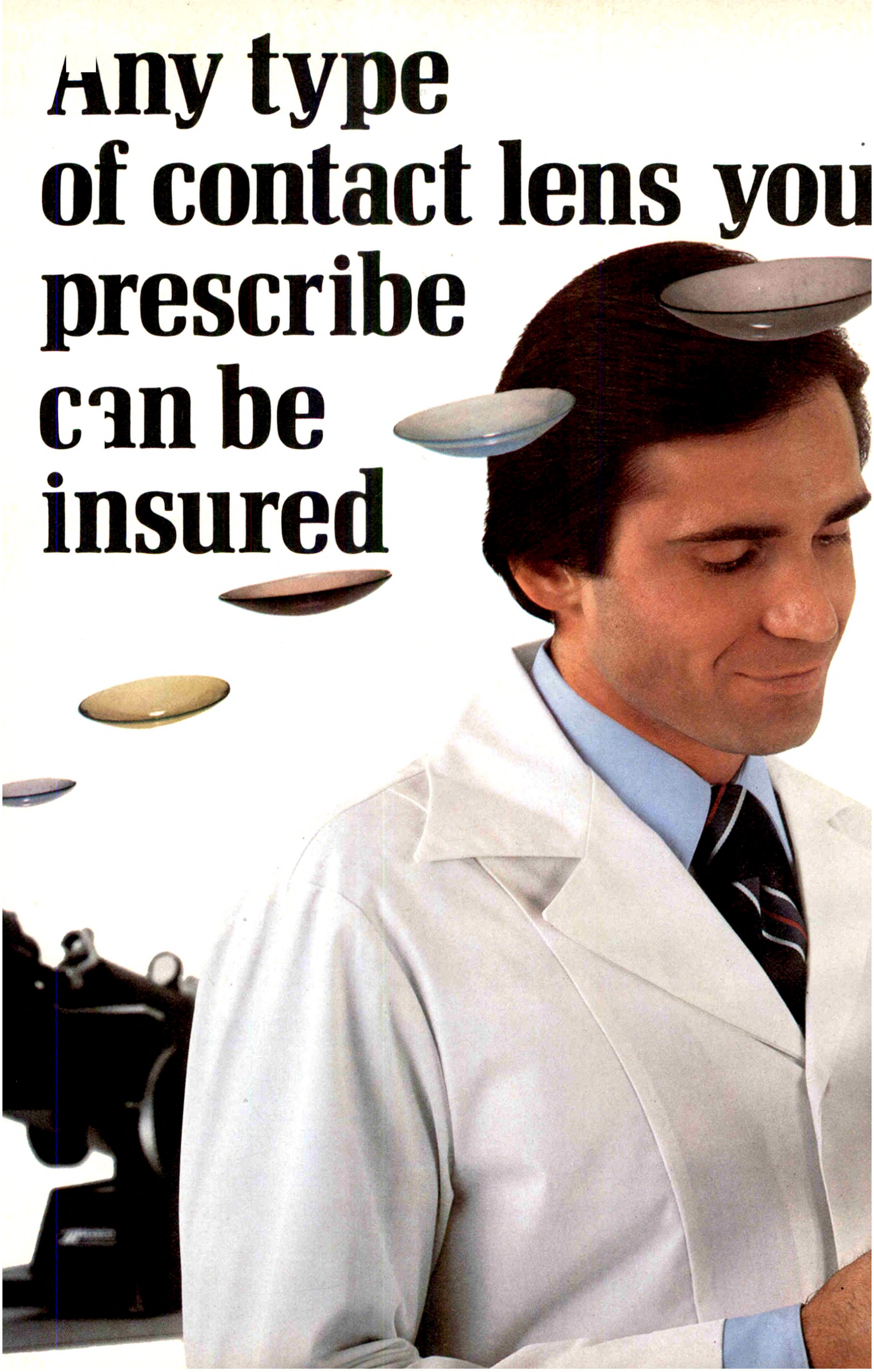
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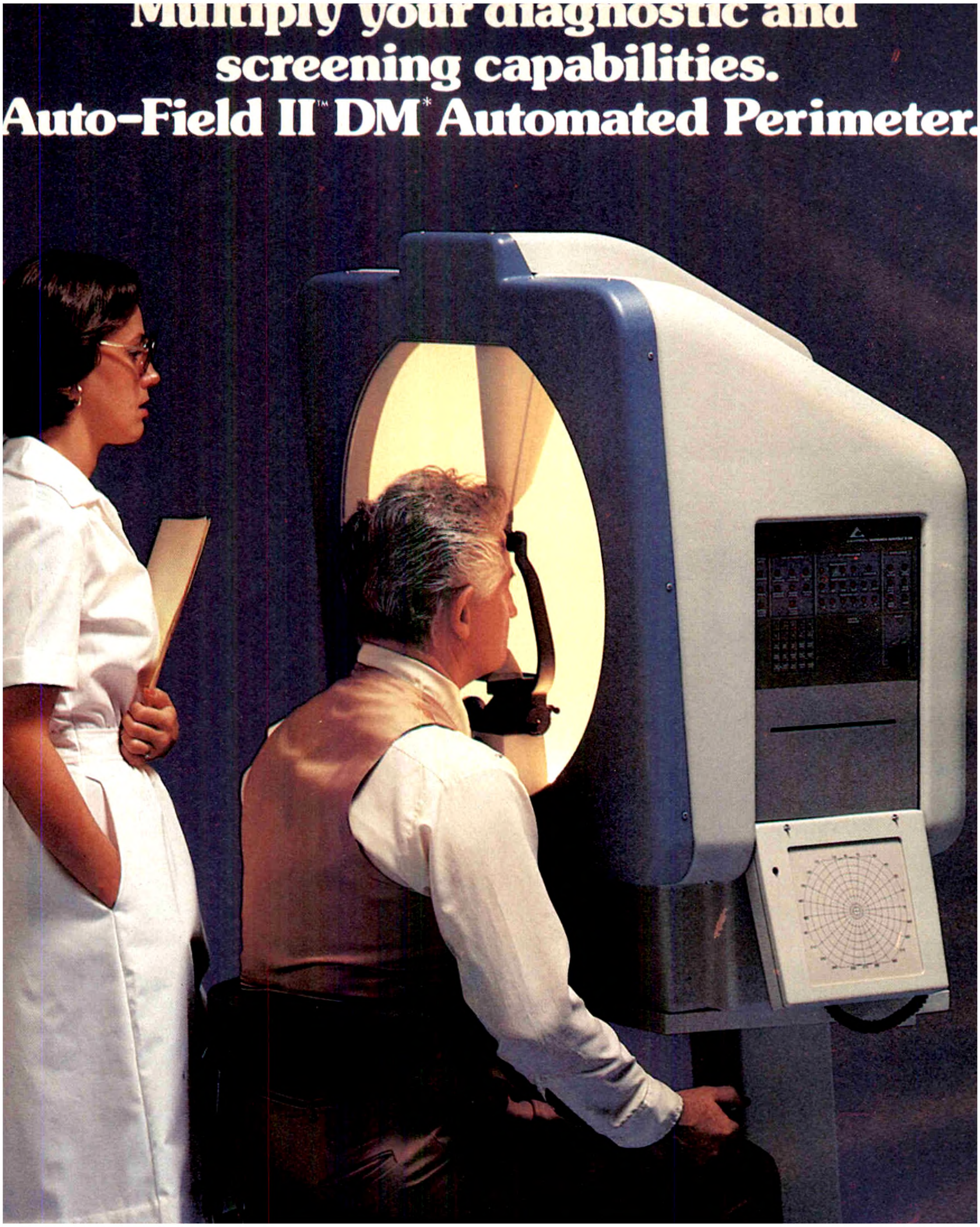

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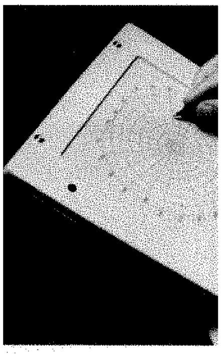

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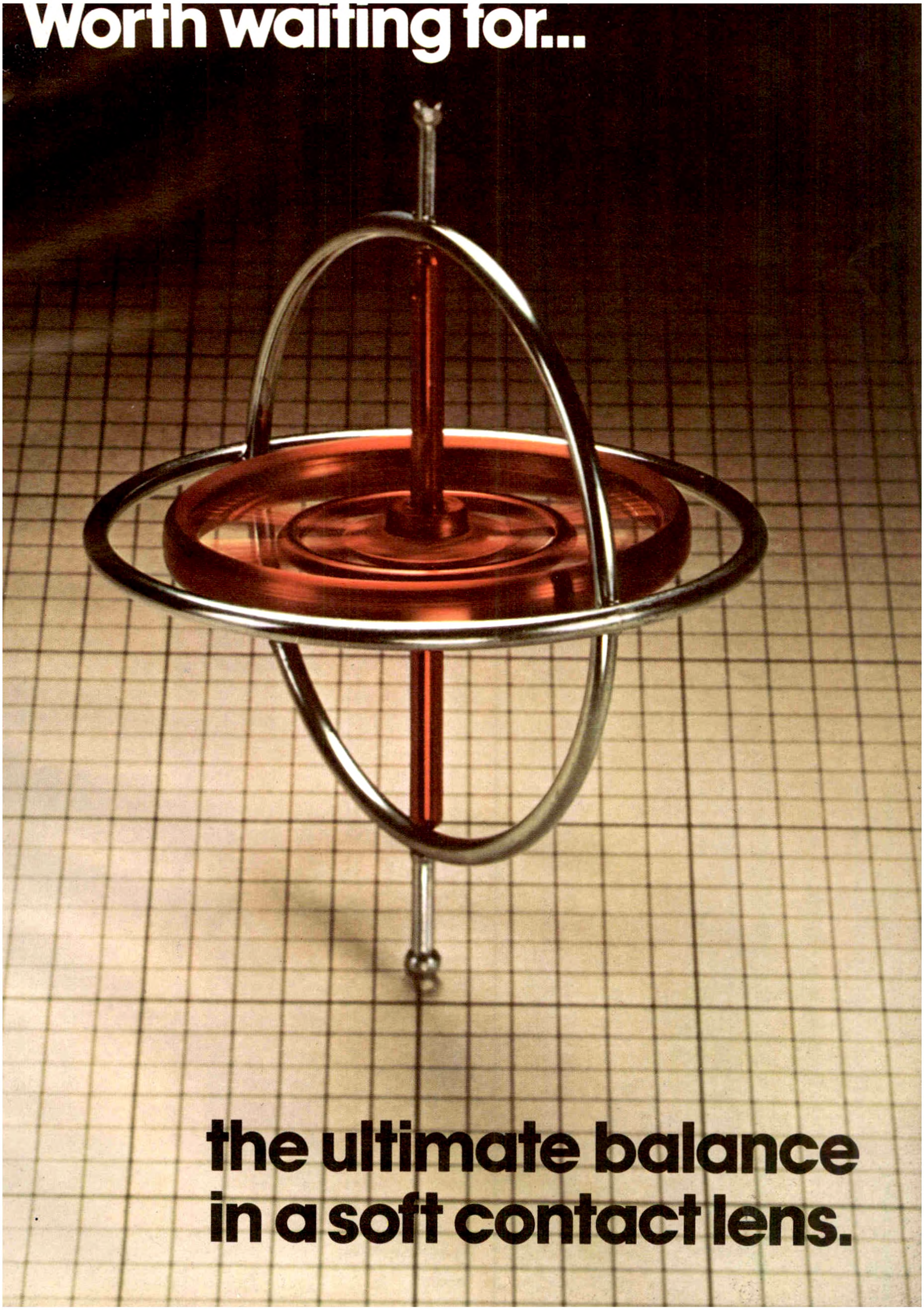
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(vifilcon A)

—the ultimate balance in performance plus comfort

Balance—the key to Softcon performance

Meets the needs of patient and practitioner.
Suits the needs of the daily, as well as occasional, wearer.

- No adaptation period is necessary.
- Immediate comfort, first day...all day...every or any day.
- Visual acuity of 20/20 or better, in 97% of patients studied.*
- Utilizes the convenient "cold" disinfection system.
- Available in a wide range of powers: minus, plus, high plus and plano.

*Data on file, Soft Contact Lens Div., American Optical Corp., Southbridge, MA 01550

Balance—the key to Softcon comfort

Lens water content of 55%.
Hydroequivalence closest to that of cornea.

- Enhances lens/cornea compatibility.
- Ensures wearer comfort without compromising handling or optical properties.
- Improves permeability and fluid exchanges.
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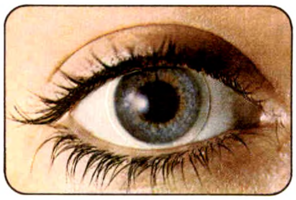

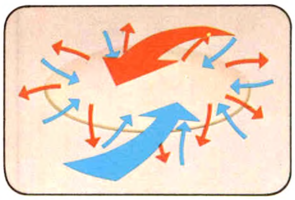

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Lathe-cut miniscleral configuration.

- Provides uniform carrier geometry for all lens powers.
- Offers "fast-fit" techniques in 6 lens geometries (3 of which will fit 80% of patients).
- Achieves superior centration and stable visual acuity.
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See next page for full prescribing information. For further details, fill out and mail this coupon.

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☐ Have representative call on ______________________
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☐ Here is my order for SOFTCON Lenses.

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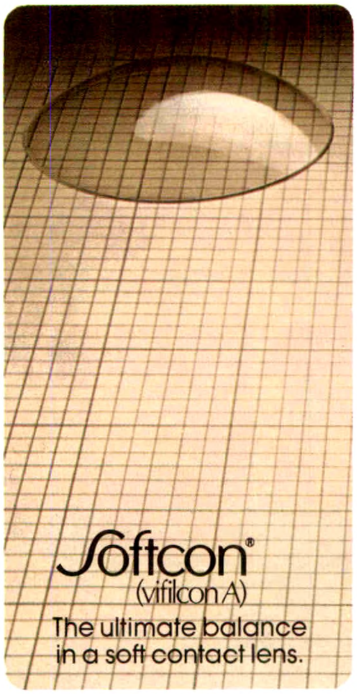

SOFTCON® (vifilcon A) Contact Lens
Vision Correction Use

DESCRIPTION The SOFTCON (vifilcon A) Contact Lens is a hemispherical flexible transparent shell which covers the cornea and part of the sclera. The lens is composed of vifilcon A, a soft hydrophilic plastic co-polymer of 2-hydroxyethyl methacrylate and povidone, USP. The chemical name is: Poly (2-hydroxyethyl methacrylate-co-ethylene dimethacrylate-co-methacrylic acid-g-povidone). When fully hydrated, the lens contains about 55% water by weight.

INDICATIONS The SOFTCON Contact Lens is indicated for the correction of visual acuity in persons with non-diseased eyes and in aphakia. It is indicated for persons who have spherical ametropias, refractive astigmatism usually 1.50 diopters or less and/or corneal astigmatism of 2.00 diopters or less.

CONTRAINDICATIONS Presence of any active infection or inflammation of the eye, insufficient tears, corneal hypoesthesia or any systemic disease which may affect the eye adversely or be exaggerated by wearing contact lenses.

WARNINGS Medications and Eye Drops—The SOFTCON Contact Lens must be stored only in FLEXSOL® soaking solution. **NEVER USE CONVENTIONAL HARD CONTACT LENS PRODUCTS WITH THE SOFTCON CONTACT LENS.** No ophthalmic solutions or medications may be used by SOFTCON Contact Lens wearers immediately prior to or during wear of the lens. No solutions other than PREFLEX®, FLEXSOL, and NORMOL® used for cleaning and chemical disinfection may be used.

Aphakic Patients—Patients who have undergone surgery for cataract removal should not be fitted with SOFTCON Contact Lenses during the immediate post-operative period; until, in the opinion of the surgeon, the eye has healed completely.

Abrasions and Infections—If a lens becomes less comfortable to the wearer than when it was first placed on the cornea, the lens should be removed immediately, and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present, or any abnormal eye condition is observed concurrently with lens wear, the lens should be removed immediately and a clinician must be consulted.

Lens Care—**WEARERS MUST ADHERE TO THE RECOMMENDED DAILY CLEANING, CARE AND DISINFECTION REGIMEN AS DESCRIBED IN THE SOFTCON WEARER'S BOOKLET. FAILURE TO ADHERE TO THE RECOMMENDED PROCEDURES MAY RESULT IN THE DEVELOPMENT OF SERIOUS EYE INFECTION.**

Wearing Restriction—The lens should be removed before sleeping or swimming, and should not be worn in the presence of irritating fumes or vapors.

Visual Blurring—If unusual visual blurring occurs, the lenses must be removed until the condition subsides. If blurring persists see your eye care practitioner.

PRECAUTIONS Storage—The SOFTCON Contact Lenses must be stored only in FLEXSOL soaking solution. If left exposed to air, the lenses will dehydrate, becoming hard and easily damaged. If a lens dehydrates, it should be carefully handled and soaked again in FLEXSOL until it returns to its normal soft, supple state.

Cleaning and Disinfection—**THE SOFTCON CONTACT LENS MUST BE BOTH CLEANED AND DISINFECTED DAILY. ONE PROCEDURE DOES NOT REPLACE THE OTHER.**

Cleaning—Daily cleaning is necessary to remove mucus and other deposits which may have accumulated on the lens surface from normal ocular secretions, airborne debris, or handling. The lenses must be cleaned before being disinfected, as deposits on the lens tend to harden and become more difficult to remove after the lenses are disinfected.

Disinfection—Lenses must be disinfected daily according to the chemical disinfection regimen. For chemical disinfection, all three solutions, PREFLEX, FLEXSOL, and NORMOL, must be used and must be changed daily. In the event that the chemical disinfection materials are not available, the lenses should not be worn.

Hygiene—Hands must be washed, rinsed thoroughly and dried with a lint-free towel before handling the lenses. COSMETICS, lotions, soaps, oils and hand creams must not come in contact with the lenses since eye irritation may result. If aerosol sprays are used while the lenses are being worn, the eyes must be kept tightly closed until the sprays have settled.

Fluorescein—Use of fluorescein while the patient is wearing the lenses may cause the lenses to become discolored. Whenever fluorescein is used, irrigate the eyes with preserved normal saline solution, and wait at least one hour before replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescein irreversibly.

ADVERSE REACTIONS Serious corneal damage may result from wearing lenses which have been soaked in conventional (hard) contact lens solutions.

Eye irritation may occur within a short time after applying a hypertonic lens. Removal of the lens will relieve the irritation. Very rarely, a lens may adhere to an eye as a result of a patient sleeping with the lens on or while wearing a hypotonic lens. If a lens adheres for any reason, apply a few drops of ADAPETTES® directly to the eye and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema as manifested by symptoms such as rainbows or halos around lights, or visual blurring may occur if lenses are worn for too long a time. Removal of the lenses and a rest period of at least one hour generally relieves these symptoms. If symptoms do not subside promptly, or if excess tearing, unusual secretions, or photophobia develop, professional consultation should be obtained.

DOSAGE AND ADMINISTRATION Fitting—Usual methods of fitting conventional (hard) contact lenses do not apply to the SOFTCON Contact Lens.

For a detailed description of the fitting technique, refer to the SOFTCON Fitting Booklet.

Wearing Schedule—The wearing schedule should be determined by the eye care practitioner. Studies have not been performed to support the safety of this lens for longer than the recommended wearing time.

Patients should wear their lenses for 8-16 hours on the day the lenses are dispensed. If patients cannot tolerate 16 hours of lens wear daily, the eyes should be examined and the lens fit re-evaluated.

Lens Care and Handling—Care must be taken on the initial visit to assure that the patient is supplied with a SOFTCON Care Kit and fully understands all proper care and handling procedures for the lenses. As with any contact lens patient, regular re-examination visits are necessary to assure ocular health and patient compliance with instructions.

How Supplied—Each lens is shipped sterile in a sealed glass vial containing buffered normal saline solution. The vial label is marked with the base curve, dioptric power, diameter and the manufacturing identification number of the lens.

Patient Care Kit—The SOFTCON Care Kit is a package required for cleaning, disinfection and storage of the lenses. It consists of the following:

- Carrying & Storage Lens Case
- PREFLEX® Sterile Cleaning Solution
- FLEXSOL® Sterile Disinfection and Storage Solution
- NORMOL® Sterile Rinsing Solution
- ADAPETTES® Sterile Lubricating Solution
- SOFTCON Wearer's Booklet

Caution: Federal law prohibits dispensing without prescription.

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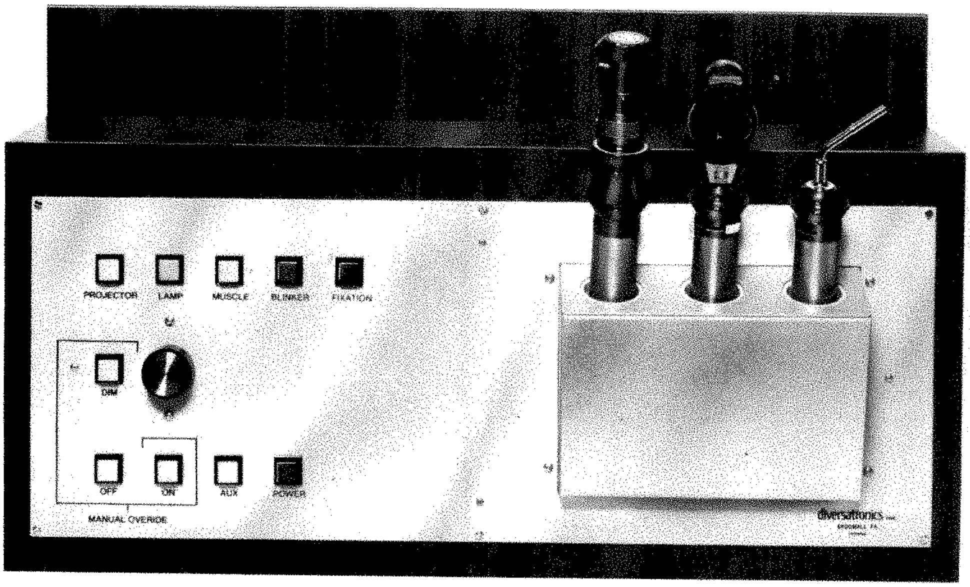

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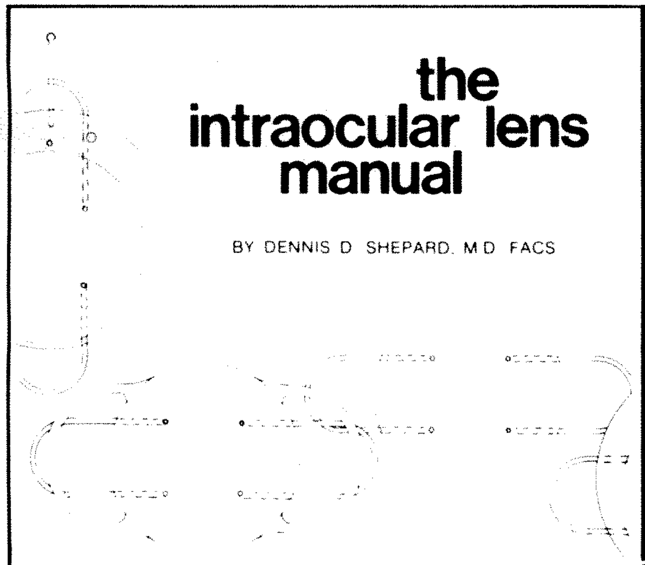
the intraocular lens manual

BY DENNIS D SHEPARD, M D FACS



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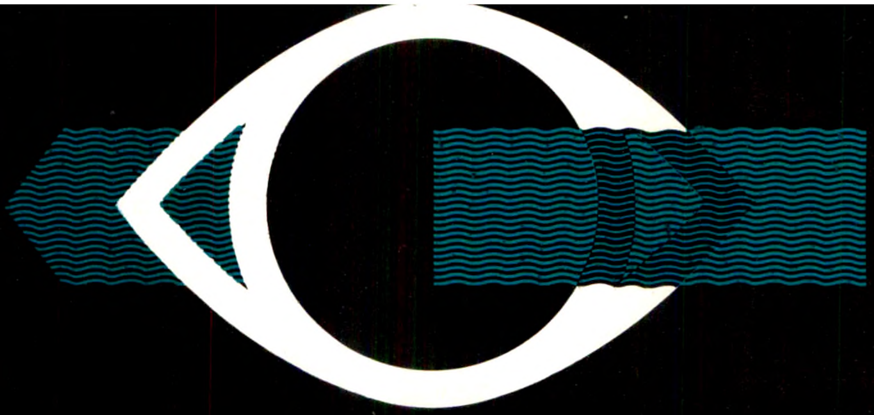

the MESO®(cabufocon) lens

Fabricated from a highly purified, medical grade of cellulose acetate butyrate, The Meso Lens offers significant advantages over both hydrogel and PMMA lenses.

The Meso Lens:

- Provides a new experience in wearing comfort without sacrificing durability or crisp visual acuity.
- Demonstrates enhanced wettability (approaching the angle produced by the normal hydrophilic cornea), improved tear exchange, and a heat-transfer coefficient considerably greater than PMMA.
- Can be fit using keratometry or conventional trial set procedures (including fluorescein patterns) for the visual correction of *astigmatism, myopia, hyperopia* and *aphakia*.

An extensive series of clinical trials on 825 wearers has demonstrated the stability, safety, and efficacy of The Meso Lens. Based on a statistical analysis of the data, it is reasonable to expect the following:

1. Rapid Adaptation/Increased Wearing Time Corneal integrity is rarely impaired. The average adaptation period is comparable to that observed with soft lenses: 8 hours wearing time within 7 days; 12 to 15 hours in less than two weeks – with the potential for interrupted-wear.

2. Reduced Signs of Overwearing The investigators cited longer wearing periods. In 1,796 slit-lamp observations, a 1.2% incidence of corneal edema and spectacle blur was reported.

3. Flexibility and Ease of Fitting Fitting The Meso Lens 0.25 diopter steeper, or with diameter differences of 0.1 or 0.2 mm, will not cause any significant change in comfort or acuity.

Parameters which affect tear exchange, such as apical clearance and intermediate bearing, are not critical. This increased latitude, particularly important in fitting astigmatic corneas, can be attributed to

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the characteristics of the cabufocon material as well as to the unique optical design of The Meso Lens.

4. Simplified Lens Care A three-solution maintenance system utilizing Preflex®, Normol®, and Flexsol® for cleaning, rinsing and disinfecting, is recommended for The Meso Lens.

Virtually no solution-related problems, such as discoloration, binding of preservatives or drug sensitivity, have been reported with The Meso Lens.

NOTE: Thermal disinfection or the use of hard lens care products is contraindicated.

5. Crisp Visual Acuity In a group of 773 eyes fit with The Meso Lens 97.3% tested 20/25 – *or better than the best possible vision attained with spectacles in the same series.*

Danker and Wohlk's exclusive molding process is utilized in the precision manufacture of The Meso Lens. The result: Uniformly good edges, high reproducibility and a smooth finish, which will resist accumulation of surface deposits.

The Meso Lens is available in a wide range of base curves and diameters, 6.0 to 9.0 mm and 6.0 to 12 mm respectively. The Meso Lens deserves a trial with new patients and wearers presenting problems with PMMA or HEMA lenses.

See the following page for prescribing information. For further details call toll free or write.

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the MESO® (cabufocon) lens

Description: The MESO® Contact Lens is a non-absorbent contact lens composed of the polymer, cellulose acetate butyrate.

It is manufactured in a range of base curves (6.0-9.0 mm) and diameters (6.0-12.5 mm) to permit custom fitting of the cornea.

Action: The MESO® (cabufocon) Contact Lens, when placed on the human cornea, acts as a refracting medium to correct myopia, hyperopia, astigmatism and aphakia.

Indication: The MESO® (cabufocon) Contact Lens is indicated for the correction of visual acuity in non-diseased eyes and aphakia.

Contra-indication: MESO® (cabufocon) Contact Lenses are contraindicated in the presence of any of the following conditions:

1. Active conjunctivitis;
2. Active or chronic trachoma infection;
3. Active herpes infection;
4. Any condition which has led to extensive corneal neovascularization;
5. Corneal hypoesthesia except that resulting from aphakia;
6. Any situation in which the wearer is unable to maintain good lens care regimen.

Warnings: The MESO® (cabufocon) Contact Lens must be stored only in Flexsol® Solution. No other storage solution is recommended. Patients must adhere to the recommended daily care regimen. Failure to do so may result in ocular infection.

If the lens becomes more uncomfortable to the wearer than when first placed on the eye, the lens should be removed, checked for dirt or scratches, and cleaned. If this condition persists or if the eye becomes red or painful, the lens should be removed immediately. If symptoms persist, your eye-care practitioner should be consulted.

The MESO® (cabufocon) Contact Lens should be removed before sleeping or swimming and should not be worn in the presence of noxious fumes or vapors. If unusual visual blurring occurs, the lens should not be worn until the condition subsides.

Precautions:

Lens Care — Storage: When the MESO® (cabufocon) Contact Lens is removed from the eye, it should be stored only in Flexsol® Sterile Soaking Solution after cleaning with Preflex®. The lens should be completely covered with the Flexsol® solution. Fill the lens case receptacle till just below the rim.

Before placing the MESO® (cabufocon) lens on the eye, rinse the lens with Normol® Solution. Discard the Flexsol® in the case, rinse the case with Normol® and dry the case. Refill with fresh Flexsol® solution each time the lens is stored.

The MESO® (cabufocon) lens, unlike the hydrophilic soft lens, does not dry out or become brittle if exposed to air. If the lens does become dry, it should be soaked in Flexsol® for a minimum of four hours before reinsertion in the eye.

Fluorescein may be used with the lens.

Lens Cleaning: Ordinary mucous and oily deposits are removed with daily cleaning of the lens with Preflex® as described in your Patient Booklet.

Adverse Reactions: As with any contact lens, corneal edema and corneal abrasions may occur if the lens is not properly fit, or if the wearing time is excessive.

Dose and Administration: A gradual build up of wearing time is recommended. A recommended schedule is as follows:

A. On the day of the initial fitting, two to four hours continuous wear.
B. Increase one hour each day for one week until eight continuous hours have been achieved.
C. If checkup is normal, wearing time is again increased by one hour a day until full time (12 or more hours) is achieved.

Wearing schedule should be determined by the eye care practitioner.

Studies have not been performed to support safety for extended wear of this lens.

How Supplied: Each lens is supplied in a sealed sterile packet. The packet is identified with the dioptric power, base curve, center thickness and manufacturer's lens number.

When the lens is removed from the packet, it should be stored in Flexsol® Solution for at least four hours prior to dispensing to ensure maximum patient comfort.

CAUTION: Federal law prohibits dispensing without prescription.

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Announcement of the
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TOPICS: Conventional hard contact lenses, hydrophilic soft gel lenses, silicone lenses, oxygen permeable hard contact lenses, aspheric, bifocal and variable focus lenses, infraocular lens implants, keratophakia and keratomileusis.

Pre-Course Basic Lectures: 4 hours on the basics of hard and soft contact lens fitting on Wednesday afternoon, March 28, 1979—Additional tuition $35.00

Basic Lectures: By Industry—no additional fee, Thursday and Friday evenings, March 29-30, 1979.

Post-Course Modification Seminar: Saturday, March 31st—8:30 a.m.—12 noon—Hard Lens Modification Course with audience participation—Limit 36 Persons—Additional tuition $35.00

For further information contact:

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CREDIT: CATEGORY I, A.M.A. PHYSICIAN'S RECOGNITION AWARD

INQUIRIES: H. MacKenzie Freeman, M.D.
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Eye Research Institute of Retina Foundation
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Tumor Evaluation and Management: fluorescein angiography, ^{32}P, ultrasonography, tumor skin testing, aqueous enzymology, enucleation—pros and cons

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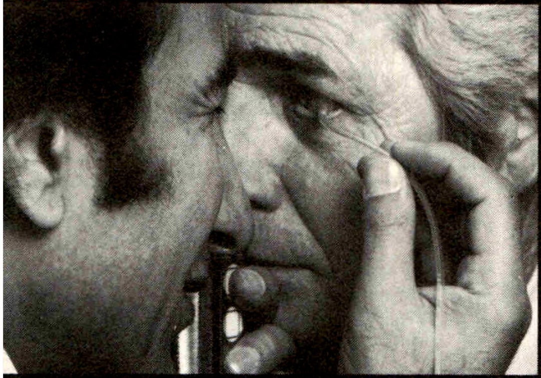

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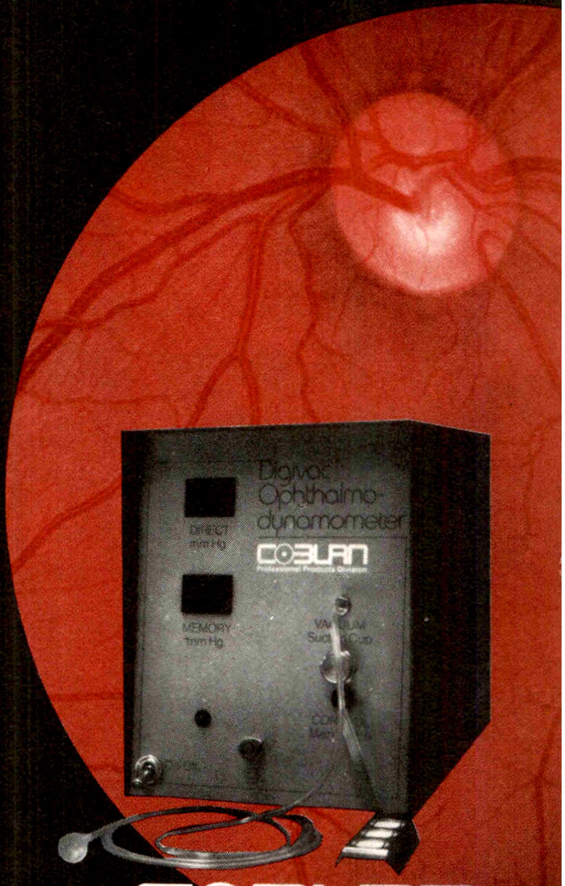

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(For full prescribing information, see package circular.)
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PHOSPHOLINE IODIDE is a long-acting cholinesterase inhibitor for topical use.

Indications: Glaucoma – Chronic open-angle glaucoma. Subacute or chronic angle-closure glaucoma after iridectomy or where surgery is refused or contraindicated. Certain non-uveitic secondary types of glaucoma, especially glaucoma following cataract surgery.

Accommodative esotropia – Concomitant esotropias with a significant accommodative component.

Contraindications: 1. Active uveal inflammation.

2. Most cases of angle-closure glaucoma, due to the possibility of increasing angle block.

3. Hypersensitivity to the active or inactive ingredients.

Warnings: 1. *Use in Pregnancy.* Safe use of anticholinesterase medications during pregnancy has not been established, nor has the absence of adverse effects on the fetus or on the respiration of the neonate.

2. Succinylcholine should be administered only with great caution, if at all, prior to or during general anesthesia to patients receiving anticholinesterase medication because of possible respiratory or cardiovascular collapse.

3. Caution should be observed in treating glaucoma with PHOSPHOLINE IODIDE in patients who are at the same time undergoing treatment with systemic anticholinesterase medications for myasthenia gravis, because of possible adverse additive effects.

Precautions: 1. Gonioscopy is recommended prior to initiation of therapy.

2. Where there is a quiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and ciliary muscle contraction that may occur.

3. While systemic effects are infrequent, proper use of the drug requires digital compression of the nasolacrimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

4. Temporary discontinuance of medication is necessary if salivation, urinary incontinence, diarrhea, profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities occur.

5. Patients receiving PHOSPHOLINE IODIDE who are exposed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin. During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable.

6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer, pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.

7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.

8. PHOSPHOLINE IODIDE (echothiophate iodide) should be used with great caution, if at all, where there is a prior history of retinal detachment.

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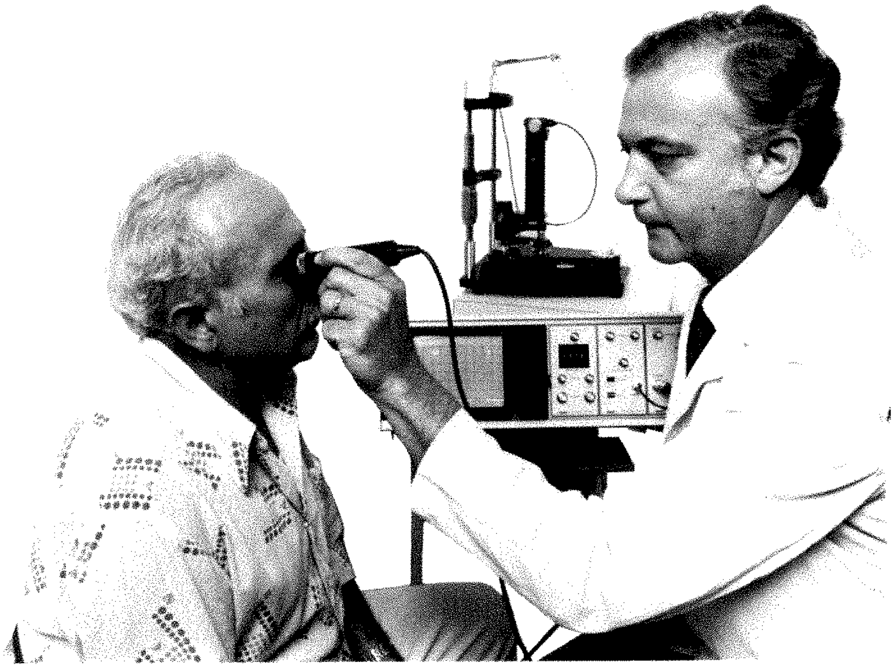

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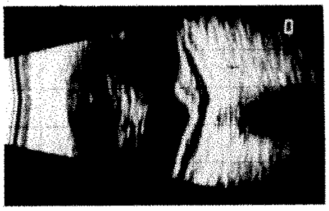

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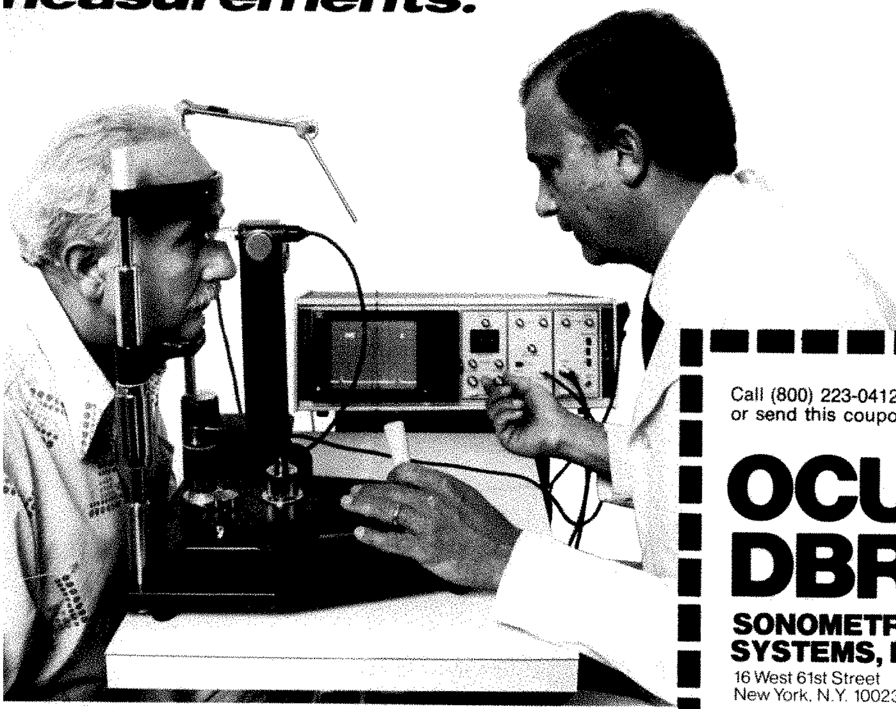

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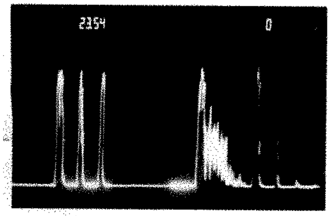

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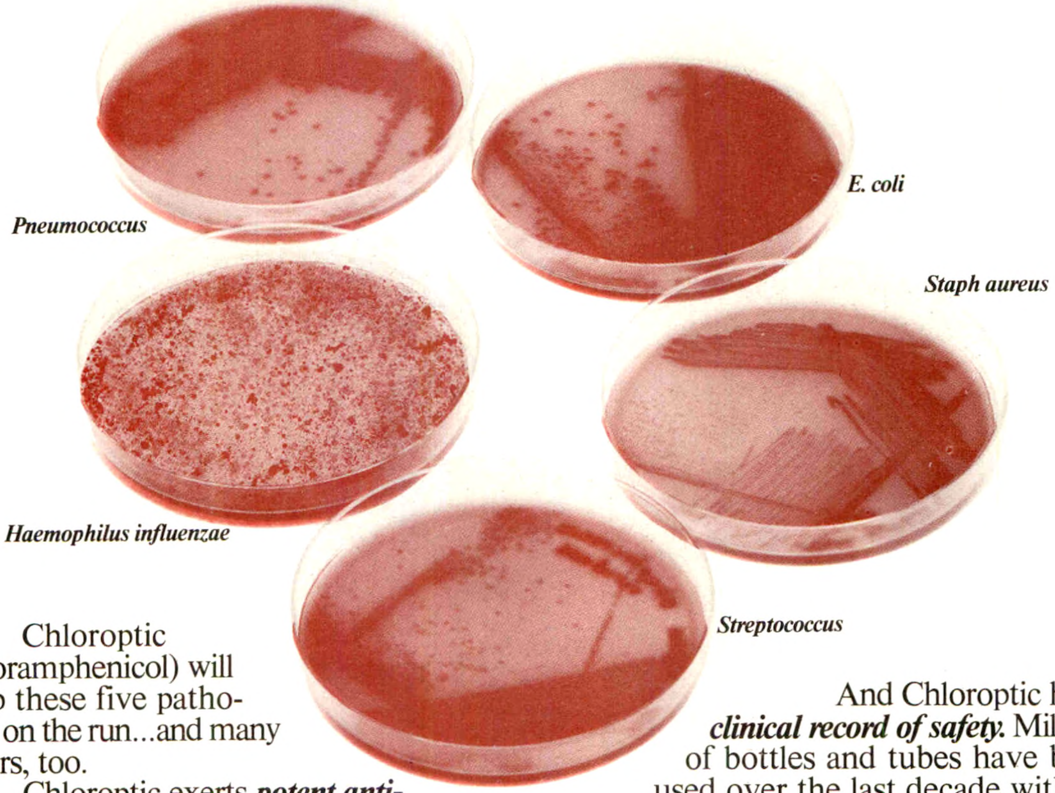

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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 87 NUMBER 2 FEBRUARY, 1979

LENS IMPLANTATION, MIOSIS, AND GLAUCOMA

Henry M. Clayman, M.D., Norman S. Jaffe, M.D., David S. Light, M.D., and Daniel M. Eichenbaum, M.D.

Miami Beach, Florida

Patients with miotic pupils resulting from prolonged miotic therapy, previous glaucoma surgery with posterior synechiae, or senile fibrosed pupillary sphincters may require cataract surgery. Because many of the intraocular lenses currently in use depend on the iris and pupillary sphincter for internal fixation and centration, some surgeons would not consider these cases suitable for lens implantation. Many patients in whom this type of surgery is considered have had glaucoma as an underlying ocular disease.

We report herein the technique and results of lens implantation and its effect on intraocular pressures in a group of patients with chronic miosis.

Methods

Several techniques have been advocated for managing the miotic pupil; these vary with implant type and each individual case.[1] The fundamental surgical principle is to perform a radial or sector iridotomy before cataract extraction, to prevent multiple sphincter ruptures. A 9-0 or 10-0 polypropylene suture is placed in the margins of the iridotomy and the cataract is extracted. The iris suture is tied, reforming the pupil, and the implant is inserted. The pupil should allow secure fixation, yet be large enough to permit atraumatic implant insertion (Fig. 1).

Results

In 1,079 intracapsular implant patients operated on between May 1974 and December 1977, 48 eyes (4.4%) had chronic miosis and required an iridoplasty; three patients had bilateral iridoplasties. The patients were observed from six to 44 months. Binkhorst iris clip and Worst Medallion implants were the only lenses used in this study. The average age of the patients requiring an iridoplasty was 76.3 years (approximately two years older than the average age of all patients); the age range was 61 to 90 years. Of the patients

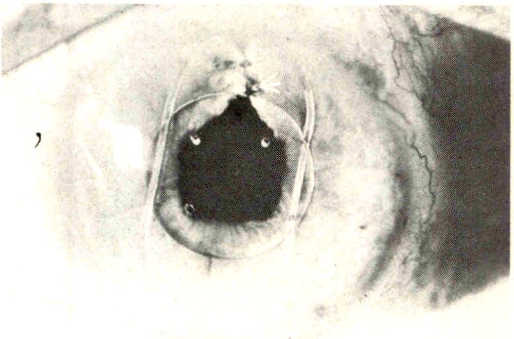

Fig. 1 (Clayman and associates). Binkhorst iris clip lens implant inserted after repair of radial iridotomy. Note iris suture (arrow).

From the Bascom Palmer Eye Institute, University of Miami School of Medicine, Miami, Florida. This study was supported by Miami Eye Foundation, Inc. Presented in part at the U.S. Intraocular Lens Symposium, March 16, 1978, Los Angeles, California.

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TABLE 1

POSTOPERATIVE VISION

Visual Acuity	No. of Eyes (%)
6/6 (20/20) - 6/12 (20/40)	43/48 (89.6)
6/15 (20/50) - 6/24 (20/80)	3/48 (6.2)
6/30 (20/100) - 6/60 (20/200)	1/48 (2.1)
Less than 6/60 (20/200)	1/48 (2.1)

TABLE 3

GLAUCOMA THERAPY

	Preoperative	Postoperative
Topical medication*	36	18
None	12	30

*Some patients also taking oral acetazolamide.

89.6% (43) achieved visual acuity of 6/12 (20/40) or better (Table 1). The causes of visual acuity less than 6/12 (20/40) were pupillary membranes (two cases), senile macular degeneration (two cases) and corneal edema (one case), secondary to increased intraocular pressure. Discission of pupillary membranes was performed on two patients, one of whom required a second discission. Vitreous was lost during cataract extraction in one case, and prophylactically aspirated before implant insertion in another.

There was little difference between average preoperative and postoperative intraocular pressures (Table 2), although 36 patients (75%) required preoperative topical glaucoma medication compared to 18 (37.5%) postoperatively (Table 3). The 12 patients (Table 3) who required no preoperative medications had either senile miosis (three cases) or a previous peripheral iridectomy with posterior synechiae, but normal intraocular pressures (nine cases). None of these patients required postoperative glaucoma treatment. Of those patients in whom postoperative glaucoma treatment was indicated, the medication was the same or less than the preoperative regimen in 83% (15) of cases (Table 4). Three patients (16.7%) had an increase in medication. The evaluation of postoperative intraocular pressures and glaucoma therapy was made at the last recorded patient visit. This averaged 16 months postoperatively.

TABLE 2

INTRAOCULAR PRESSURES

mm Hg	Preoperative, No. of Eyes	Postoperative, No. of Eyes
11 - 15	12	14
16 - 20	27	26
21 - 25	8	7
26 - 30	1	0
31	0	1

DISCUSSION

The patients studied either had a diagnosis of glaucoma from the operating surgeons, or from another ophthalmologist. In many of the patients the opaque media and miosis prevented reliable visual field testing and evaluation of the optic nerve, and therefore the diagnosis of concomitant glaucoma may be questioned.

The results show that cataract extraction with lens implantation did not have a deleterious effect on intraocular pressures, and control of postoperative intraocular pressures was either easier or not required in most patients.

TABLE 4

POSTOPERATIVE GLAUCOMA THERAPY (18/48 EYES)

	No. of Eyes (%)
Less than preoperative	8/18 (44.4)
Same as preoperative	7/18 (38.9)
More than preoperative	3/18 (16.7)

There are two important qualifications to this statement. First, many clinical observations have been made regarding the effects of cataract extraction on intraocular pressure and pre-existent glaucoma.[2,3] Removal of the crystalline lens often has a beneficial effect on intraocular pressures, although this may be temporary. Whether the intraocular pressures will eventually rise or be difficult to control, or both, in these patients is not known, but lens implantation does not negate the ocular hypotensive effects of cataract extraction alone.

The second qualification is the selection of patients. Only one of the patients in this series had a preoperative pressure greater than 25 mm Hg; this was the patient whose postoperative pressure was greater than 31 mm Hg, and who had postoperative corneal edema. The other patients had more easily controlled intraocular pressures. We therefore screened the patients preoperatively, and with one exception, did not perform implant surgery on severe glaucoma patients with marginal control.

SUMMARY

In a series of 1,079 patients, average age 76.3 years, 48 eyes required an iridoplasty for chronic miosis and, of these, visual acuity was 6/12 (20/40) or better postoperatively in 89.6% (43). In those patients with glaucoma, the control of intraocular pressure was the same or better than postoperative levels in 83.3% (15) of patients. Lens implantation was not contraindicated in the miotic patient if adequate iris surgery was performed before both cataract extraction and implant insertion. Where there was concomitant glaucoma, lens implantation was likewise not contraindicated if the glaucoma was well controlled.

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CATARACT EXTRACTION IN GLAUCOMA

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New York, New York

When cataract surgery is necessary in a glaucomatous patient,[1-4] many surgeons advocate simple lens extraction. Normalization of intraocular pressure may occur particularly if fine, buried sutures are used, implying some type of filtration.[5] Hyposecretion, has also been implicated as the mechanism for pressure normalization when no other obvious explanation is apparent.[5]

More predictable results than those obtained by cataract extraction alone occur with combined procedures,[6-13] but are associated with greater surgical risk and are not universally recommended.

The data of published studies of the effect of cataract extraction or combined procedures on intraocular pressure generally have been pooled and do not have adequate controls. We report herein on a combined procedure, either cyclodialysis cataract extraction or cyclodialysis canalicular trabeculectomy cataract extraction, performed in one eye of glaucomatous patients, and a standard procedure performed in the second equally glaucomatous eye, in order to determine the physiology of intraocular pressure control after cataract extraction, as well as which surgical approach is more appropriate.

Material and Methods

We performed combined surgery on 120 patients who have been observed for at least two years. All patients required cataract extraction for visual purposes and all but two patients had open-angle glaucoma with field and disk changes. Two patients had previous peripheral iridectomies for angle-closure glaucoma and required miotics. They also had marked field changes. Forty-seven patients were suitable for this present study, as they had reasonably similar bilateral glaucoma and cataracts. Twenty-four patients had undergone a combined procedure in one eye and simple cataract extraction in the other. Twenty-three patients have had surgery to one eye; the fellow eye has either not been operated on or been observed for two years. Sixteen of the eyes undergoing a combined procedure would have required glaucoma surgery if no cataract were present because of inadequate glaucoma control. The remaining eight, though needing cataract surgery, were well controlled. In the fellow control eyes, 14 had inadequate glaucoma control and ten were well controlled.

The 24 combined procedures were equally divided among cyclodialysis cataract extraction and cyclodialysis canalicular trabeculectomy cataract extraction. The ages of both groups ranged from 50 to 87 years. The follow-up period was from five to 12 years in the cyclodialysis group, two to four years in the cyclodialysis canalicular trabeculectomy group, and from three to ten years in the cataract extraction control group.

Though both eyes were similar in all cases, the combined procedure was always performed in the eye with the more severe glaucoma, as evidenced by higher pressure, more severe field loss, or greater cupping.

Surgery was usually delayed in either

From the Department of Ophthalmology of the New York Medical College (Drs. Galin and Hung), and the Department of Ophthalmology, Mt. Sinai Medical Center, (Dr. Obstbaum) New York, New York. This study was supported by grants from the Ophthalmological Foundation of America, Inc., and by United States Public Health Service International Research Fellowship F052080.

Reprint requests to Miles A. Galin, M.D., Department of Ophthalmology, New York Medical College, 1249 Fifth Ave., New York, NY 10029.

eye until the patient was visually symptomatic or glaucoma control was inadequate. Attributing field loss to glaucoma rather than to further reduction in visual acuity is not simple, and frequent kinetic, and static fields were performed.

Surgery in all control cases involved a limbal based conjunctival flap, a vertical groove, preplaced 6-0 silk sutures, a biplaned incision, peripheral iridotomies, synechiotomy when necessary, and lens extraction. This series was not complicated by vitreous loss, wound leakage, or any other complication.

In the cyclodialysis lens extraction procedure, cyclodialysis was done after groove formation and suture placement, as described previously.[14] No anterior segment complications occurred though one patient had a choroidal hemorrhage, which occurred at the time of surgery and subsequent secondary vitreous hemorrhage. This took months to clear but resulted in visual acuity of 6/6 (20/20). Cyclodialysis canalicular trabeculectomy cataract extraction altered the lens extraction only slightly. A 3 × 3- to 4-mm scleral flap 0.25 to 0.5 mm deep was made at 12 o'clock (Fig. 1). The anterior extent of the flap ended at the surgical corneoscleral limbus and a limbal groove with preplaced 6-0 sutures was created laterally and medially, as for routine cataract extraction (Fig. 2). The posterior scleral flap was excised (Fig. 3, top), the eye opened with scissors, and iridotomies performed (Fig. 3, bottom). Since the deep scleral flap was created posterior to the scleral spur, a cyclodialysis done under total visual control, slightly larger than the 3-mm width of the scleral flap[15] resulted. After the cataract was removed, the flap site was closed with two interrupted 8-0 silk sutures, and the remainder of the wound closed routinely.

One anterior chamber complication occurred in the cyclodialysis canalicular trabeculectomy lens extraction procedure group. A hyphema developed on the third day which nearly progressed to an eightball hemorrhage. The clot was evacuated, and the patient's postoperative course

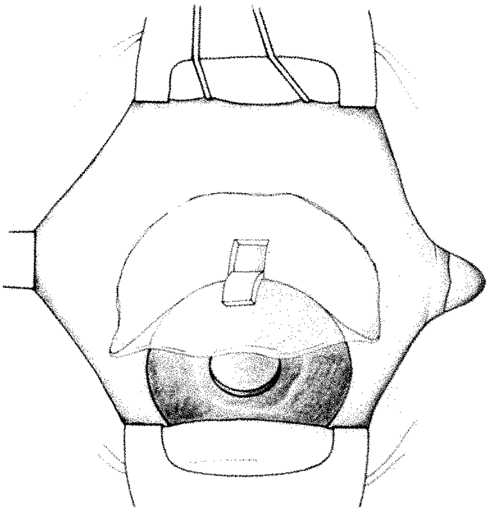

Fig. 1 (Galin, Hung, and Obstbaum). After a limbal based conjunctival flap is created, a 3 × 3- to 4-mm limbal based lamellar scleral flap is made.

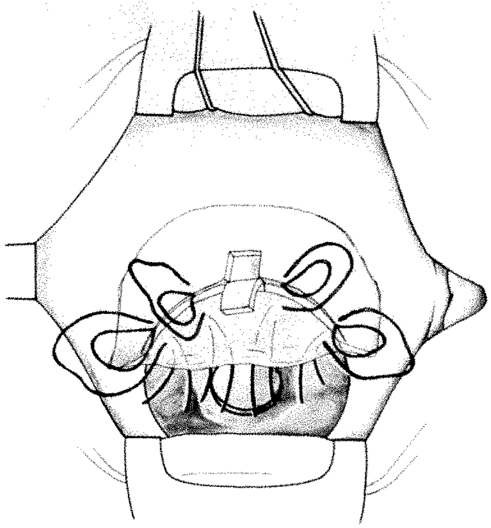

Fig. 2 (Galin, Hung, and Obstbaum). A standard limbal groove is created medial and lateral to the lamellar scleral flap and threaded with preplaced sutures.

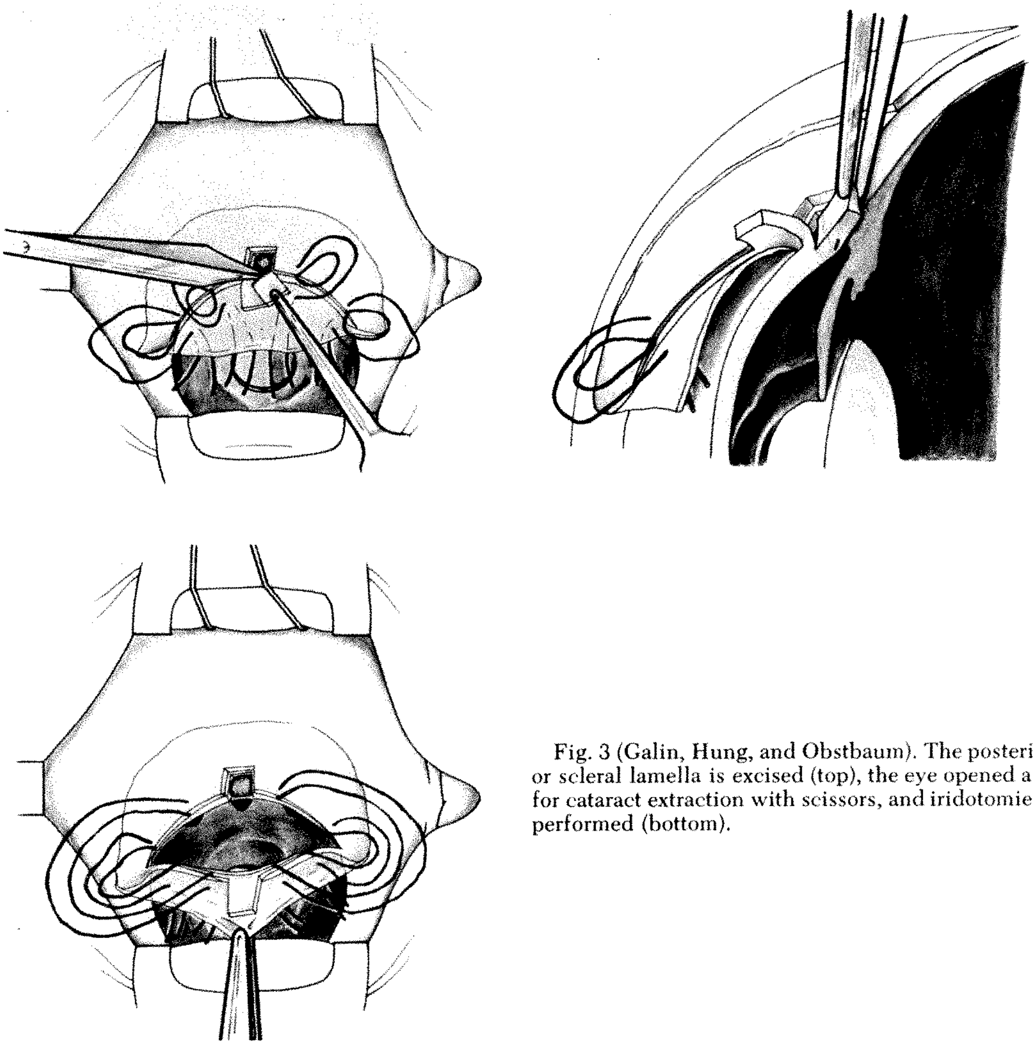

Fig. 3 (Galin, Hung, and Obstbaum). The posterior scleral lamella is excised (top), the eye opened as for cataract extraction with scissors, and iridotomies performed (bottom).

was uneventful. No obvious cause of the bleeding was apparent at the initial surgery or subsequent intervention.

RESULTS

Visual acuity was 6/12 (20/40) or better in 20 control eyes. Visual acuity was less than this level in four cases, three from advanced glaucoma, and one from a macular lesion. In the combined procedure group, visual acuity was 6/12 (20/40) or better in 18 eyes. As in controls, the cause of reduced visual acuity was glaucoma in four cases and macular degeneration in two. In the cyclodialysis lens extraction group, six of 12 eyes required no medication. The other combined group had nine of 12 patients free of medication. The cutoff point on no medication was arbitrarily set at a diurnal curve that did not exceed 18 mm Hg.

In this group without medication, no progression of glaucomatous visual field loss or further disk changes occurred. Besides the previous complication mentioned in the cyclodialysis lens extraction group, one patient had a large choroidal detachment and two had small hyphemas.

No therapy was given to any of these patients, and all absorbed the transudate or blood without difficulty. No other complication occurred in the cyclodialysis canalicular trabeculectomy cataract extraction group.

In the cataract extraction group, two eyes required no further medication by using the above criteria. All others required medication. There were no surgical or postoperative problems in any case. One of the two cures in the control group had a significantly improved outflow facility, and the other did not. No blebs and no clefts were present, and a rise in intraocular pressure on perilimbal suction occurred in both cases.

In the successful cyclodialysis cataract extraction group, a cleft was visible in each instance and perilimbal suction cup analysis resulted in a fall in intraocular pressure.[16] Clefts were visible in two of the remaining six patients, but functional analysis by perilimbal suction induced a rise in intraocular pressure in each instance. Gross bleb formation was present in six of nine successful cyclodialysis canalicular trabeculectomy cases and clefts visible in three, two with blebs and one without. Perilimbal suction cup analysis was impossible to perform adequately because of the bleb area in this group.

DISCUSSION

The evaluation of the effect of cataract extraction on glaucoma control has been expressed as changes in mean intraocular pressure or alteration in glaucoma medication. More meaningful criteria that have been suggested are the number of patients that require no medication or have required further glaucoma surgery.[5] These criteria have been reserved primarily in cases where the glaucoma was uncontrolled preoperatively and after surgery. The present series cannot be analyzed by the latter standard, and though the series is too small for statistical analysis, there is little question that the combined procedures were more predictable and more consistent, despite a success rate of only 50 to 75% without medication. Every effort was used to assure good cataract wound closure in both series, and a two-planed incision closed with multiple 6-0 silk sutures rarely results in serious leakage.

The choice of operation for cataract coexisting with open-angle glaucoma has historically varied, depending for the most part, on the status of glaucoma control. In well-controlled cases, most surgeons will perform cataract extraction alone.[17] For inadequately controlled glaucoma, it had been common to perform glaucoma surgery as an initial procedure.[18-20] However, subsequent surgical difficulties at the time of cataract surgery and difficulties in the preservation of bleb function were discouraging.[21] These problems, coupled with better surgical technique, have encouraged the development of combined procedures.[22] A further stimulus to combined surgery is the observation that glaucoma surgery in eyes with existing immature cataracts often enhances cataract maturation.

We are presently performing a modified cyclodialysis canalicular trabeculectomy cataract extraction for patients with cataract and glaucoma in which the cyclodialysis is remarkably larger than the width of the lamellar scleral flap. Since the trabeculectomy flap is initiated posterior to the scleral spur, a cyclodialysis under direct visualization is performed as part of the procedure in order to disinsert the scleral spur. The disinsertion is extended laterally and medially to create at least a 30-degree cyclodialysis (Fig. 4). These maneuvers do not lead to significant hyphema, nor does the initial scleral spur disinsertion, as evidenced by the rare occurrence of hyphema after such operations in phakic eyes. Usually hyphema after cyclodialysis is caused by tearing

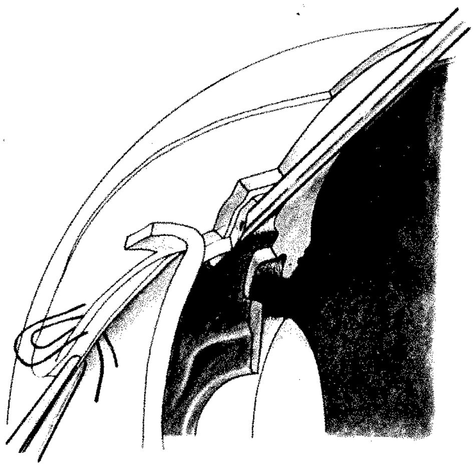

Fig. 4 (Galin, Hung, and Obstbaum). Cyclodialysis canalicular trabeculectomy cataract extraction in which the cyclodialysis is approximately 30 degrees.

of ciliary muscle by the cyclodialysis spatula as one blindly attempts to disinsert this muscle at its attachment. This is frequent in the aphakic eye where the ciliary body is not under traction. When performing the cyclodialysis as part of a trabeculectomy, the surgeon sees the attachment of the ciliary body and should be able to disinsert it cleanly. Though loose closure of the lamellar scleral section is desirable in the phakic eye to assure filtration, a firm closure is necessary in the combined procedure to assure a deep chamber. Filtration will occur in a moderate percentage of cases and the additional controlled cyclodialysis results in a highly successful procedure.

Long-term follow-up studies for simple cataract surgery in glaucomatous patients are rare. Balanced against a predictable, reproducible success rate of at least 50% with combined procedures, simple cataract extraction would not be indicated even for cases of well-controlled glaucoma, were it not for the increased morbidity of combined procedures. The procedure of cyclodialysis canalicular trabeculectomy cataract extraction combines the most favorable features of each procedure while reducing the risk components. The cyclodialysis is carried out under direct visualization, reducing the potential for bleeding, and the lamellar scleral flap is reasonably well closed, reducing the potential for a flat chamber. The ease of performing lens extraction remains unaltered. We believe more extensive application of this procedure should be used in combined disease.

SUMMARY

Cyclodialysis lens extraction or cyclodialysis canalicular trabeculectomy lens extraction in one eye and cataract extraction in the fellow eye were performed in 24 glaucoma patients. The combined procedure follow-up was from five to 12 years in the first group, two to four years in the second group, and from three to ten years in the control group.

The 50% success rate of cyclodialysis lens extraction can be enhanced with no significant increase in surgical morbidity or postoperative complications by performing a cyclodialysis canalicular trabeculectomy where the disinsertion of the scleral spur is carried out under direct visualization.

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TRANSFER OF BACTERIAL INFECTION BY DONOR CORNEA IN PENETRATING KERATOPLASTY

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AND
Rudolph M. Franklin, M.D.
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The proper selection of donor material for keratoplasty plays an important role in both the immediate and long-term postoperative period. The criteria for selection of donor material for keratoplasty are somewhat arbitrary. Generally accepted bases for excluding donor material include corneal deformities and scars, vascularization, endothelial damage, retinoblastoma, and hepatitis.[1] Although corneal material from donors with a variety of probable or possible systemic infections is regarded by some as unsuitable for transplantation,[2] this view is not universally accepted.[3] Little evidence has been presented to indicate transfer of infection with donor corneas except in one possible instance of a fatal slow-virus infection,[4] a case of cryptococcal endophthalmitis,[5] and several cases of bacterial endophthalmitis.[6,7]

We report herein on a fulminating bacterial endophthalmitis that occurred in two recipients from the same donor after penetrating keratoplasty. The donor died with Hodgkin's disease complicated by peritonitis and possible septicemia. Although the donor corneas appeared to be normal on both gross and slit-lamp examination, we think the donor material was heavily infected at the time of operation.

Case reports

A 45-year-old man with Hodgkin's disease developed a board-like abdomen and high fever. Laparotomy showed four areas of jejunal perforation, and the patient died the next day. Cultures of the donor's abdominal cavity exudate obtained at the time of laparotomy showed *Escherichia coli.* His eyes were obtained two hours after death and were used six hours later for corneal transplantation to the recipients described in this report. Slit-lamp examination of both eyes before transplantation showed no abnormality.

The first recipient was a 26-year-old man with advanced keratoconus and visual acuity of counting fingers at 1.5 m in his right eye and 1 m in the left. He underwent an 8.5-mm penetrating keratoplasty of the left eye. This was the first of five consecutive keratoplasties performed by the same surgeon (A.A.K.) that evening.

The procedure was performed under local anesthesia, using the operating microscope. After the donor material had been rinsed with chloramphenicol 1% and sulfacetamide solution 10%, the epithelium was removed and the corneal button sutured to the recipient bed with running 10-0 monofilament nylon. The recipient eye was treated topically with chloramphenicol 1% and sulfacetamide 10%, homatropine 2%, and tetracycline ointment.

The next morning the patient complained of pain in the operated eye. On examination, moderate eyelid edema, purulent discharge, and severe conjunctival chemosis were noted. The donor cornea showed diffuse and deep white infiltrates with patches of necrosis, and a hypopyon was present in the anterior chamber. Visual acuity was light perception. Smears of the exudate showed gram-negative rods and a few gram-positive cocci. The patient was given appropriate systemic and topical antibiotics. Cultures reported 24 hours later showed heavy growth of *Pseudomonas aeruginosa* and no other organisms.

The graft became totally necrotic with wound dehiscence and iris prolapse 24 hours after operation. Thirty-six hours after the initial operation, the graft was removed. Because of a dense white reflex, the lens was extracted, followed by a spontaneous loss of purulent vitreous. About 80% of the purulent contents of the vitreous cavity was then removed surgically, and a second corneal allograft sutured in place.

Postoperatively, the graft remained clear and the conjunctival reaction gradually subsided. However, the patient lost light perception three days after the

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operation, and the eye became phthisical within three weeks.

The second recipient was a 42-year-old man who received a 7-mm full-thickness graft to his left eye for a partially vascularized central leukoma of old herpetic keratitis. The preparation and operative technique were the same as for the first recipient.

About ten hours after operation, the patient complained of pain in the operated eye. On examination, visual acuity was light perception. There was moderate purulent discharge, the conjunctiva was chemotic, and the graft was totally hazy with scattered necrotic patches. An intense anterior chamber reaction was present, and no red reflex was seen. Smears taken at this time showed gram-negative rods and several gram-positive cocci. Antibiotic therapy was initiated and cultures subsequently showed heavy growth of *P. aeruginosa*, with sensitivity only to carbenicillin. Twenty-four hours after the operation the graft showed areas of necrosis with wound dehiscence and iris prolapse. Regrafting was done 36 hours after the first operation. Fibrin and purulent material were irrigated from the anterior chamber, but the lens was clear and a pale orange reflex was seen.

Postoperatively, antibiotics were continued for ten days. The graft remained clear, anterior chamber and adnexal reaction gradually subsided, and the patient was discharged from the hospital with a visual acuity of 6/15 (20/50) with pinhole. Three weeks postoperatively, the anterior chamber and vitreous reaction had subsided and visual acuity had improved to 6/9 (20/30).

During the evening of the initial corneal transplantation of these two recipients, three other patients also received corneal allografts. The donor material for all five patients had been maintained under similar conditions, but these other three allograft recipients had uneventful postoperative recoveries.

DISCUSSION

The two recipients had fulminating endophthalmitis after penetrating keratoplasty with material from the same donor. The following evidence suggests transmission of infection from the donor corneal material: (1) signs of intense inflammation appeared within hours after operation in both cases; (2) postoperative intense inflammation was clinically evident within the full thickness of donor cornea; (3) the operative procedures performed on these patients were two out of five operations performed in one evening, but not in succession, and the other three patients had an uneventful postoperative course; (4) the same type of organism was recovered in both cases; (5) the donor of the allografts died of a chronic illness complicated terminally by peritonitis and probably a gram-negative septicemia.

Although gross and slit-lamp examination of the donor material before operation showed no sign of abnormality of the corneas, the clinical course in both cases suggests that the corneas were infected with microorganisms. Possibly the debilitated state of the donor prevented any discernible reaction against the infection, and thus the corneal tissues appeared normal. However, after transplantation the recipient eye developed a full inflammatory reaction against the organisms in the infected tissue.

Whether the *Pseudomonas* infection of the donor corneas was a result of bacteremia or local invasion of ocular tissues is not certain. Blood cultures were not obtained from the donor. A recent study of eyes obtained from patients harboring systemic infections showed that seven of 20 corneas were infected, along with positive aqueous culture in three of those seven, whereas none of 25 other corneas was infected when obtained from nonseptic donors.[8] That study fails to establish conclusively the source of the bacteria, but it does suggest a systemic origin of the bacteria rather than invasion from the corneal or conjunctival surface.

It seems doubtful that soaking donor material in antibiotic-enriched medium would eliminate all donor infections.[8] Our cases, as well as several others,[4-7] suggest caution in the use of donor material obtained from patients whose death was associated with infection. Continued scrutiny of donor material, and an awareness that infection can be transferred with corneal allografts, may help to prevent recipient morbidity.

SUMMARY

A 45-year-old man died of Hodgkin's disease complicated by peritonitis and

possible septicemia. His corneas were used for transplant in a 26-year-old man with advanced keratoconus and a 42-year-old man with vascularized central leukoma of old herpetic keratitis. Both recipients developed a fulminating endophthalmitis with *Pseudomonas aeruginosa.* We believe that the donor corneas, although clinically normal, were heavily infected, with signs of inflammation possibly suppressed by the Hodgkin's disease.

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THE CLINICAL COURSE OF SERPIGINOUS CHOROIDOPATHY

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Serpiginous choroidopathy is a progressive condition that primarily affects the choriocapillaris and retinal pigment epithelium and often leads to profound visual loss if the foveal or parafoveal areas are involved. It is almost always bilateral and thus far has been described only in whites, although we have seen two cases in blacks. The cause remains obscure.

The clinical appearance and fluorescein angiographic findings in serpiginous choroidopathy have been well described.[1-3] The long-term follow-up and clinical course of this progressive disease, however, have not been well documented, particularly with regard to the prognosis for central vision. Chisholm, Gass, and Hutton[4] provided the only available information in their series of 40 eyes observed from single visits to nine years. A significant reduction in visual acuity was seen in only three eyes of their series, although many started out with poor vision. We report herein the clinical course of this disease, particularly with regard to visual acuity.

SUBJECTS AND METHODS

Of the nine patients in our study, seven were men and two were women. Their average age was 46 years with a range of 22 to 58 years. All the patients were bilaterally affected except for one patient who lost an eye in childhood because of trauma, so that there was a total of 17 eyes in our study. The period of follow-up ranged from two to ten years.

We reviewed all patients with serpiginous choroidopathy seen here who had a minimum of two years follow-up. Nine patients met this criteria. All patients had multiple fundus photographs and fluorescein angiograms, and had been seen by us on multiple occasions for clinical examination. Eight out of nine patients were recalled, and detailed ocular, systemic, and social histories were taken together with complete ocular examination, indirect ophthalmoscopy, and slit-lamp biomicroscopy with a fundus contact lens. Fundus photographs and fluorescein angiograms were taken. Electroretinography and electro-oculography as well as visual fields were performed.

CASE REPORTS

Case 1—A healthy 48-year-old white man noticed painless decreased vision in his right eye in September 1966. His past medical and family history were unremarkable. The patient had worked in a plastic plant since 1959, and had been exposed constantly to asbestos, phenol, phenole resins, furfuryl, furene resins, paraformaldehyde, hydrochloric acid, formaldehyde, epoxy resins, silica fibers, polypropylene, polyethylene, and polycarbonate.

From the Kresge Eye Institute, Wayne State University, Detroit, Michigan (Dr. Weiss and Mr. Christopherson); and the Retina Service, Wills Eye Hospital, Philadelphia, Pennsylvania (Drs. Annesley and Shields, and Mr. Tomer). This study was supported in part by the Retina Research and Development Foundation, Philadelphia, and the Lions Club of Pennsylvania. Dr. Weiss was a Heed Foundation Fellow (1975-1976).

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On examination Sept. 9, 1966, best corrected visual acuity was R.E.: 6/24 (20/80) and L.E.: 6/6 (20/20). External and slit-lamp examinations were normal. The right fundus showed a gray discoloration of the pigment epithelium on the nasal edge of the fovea, and the left fundus was normal. Visual acuity in the right eye gradually improved over the next six months to 6/6 (20/20). At the onset of his visual loss, the patient was given oral prednisone, 40 mg/day for three months, after which the dosage was gradually tapered and discontinued.

Four years later visual acuity in his left eye decreased to 6/60 (20/200). Examination revealed a typical active area of a gray discoloration of the pigment epithelium. In contrast to the right eye, the active area involved the whole fovea, rather than just its margin. Visual acuity in the left eye remained at 6/60 (20/200) for seven years.

Since May 1970, the patient had experienced no change in vision and although his visual acuity remained stable at 6/6 (20/20) in the right eye and 6/60 (20/200) in the left eye, his disease continued to progress (Fig. 1). The asymptomatic progression of the disease was not recognized until the serial fundus photographs were reviewed, which showed centripetal progression from the fovea toward the disk.

The disease was characterized by periods of activity alternating with periods of quiescence. In the active stage, a localized area of gray-green or cream-colored opacification developed beneath the retina at the level of the retinal pigment epithelium. The

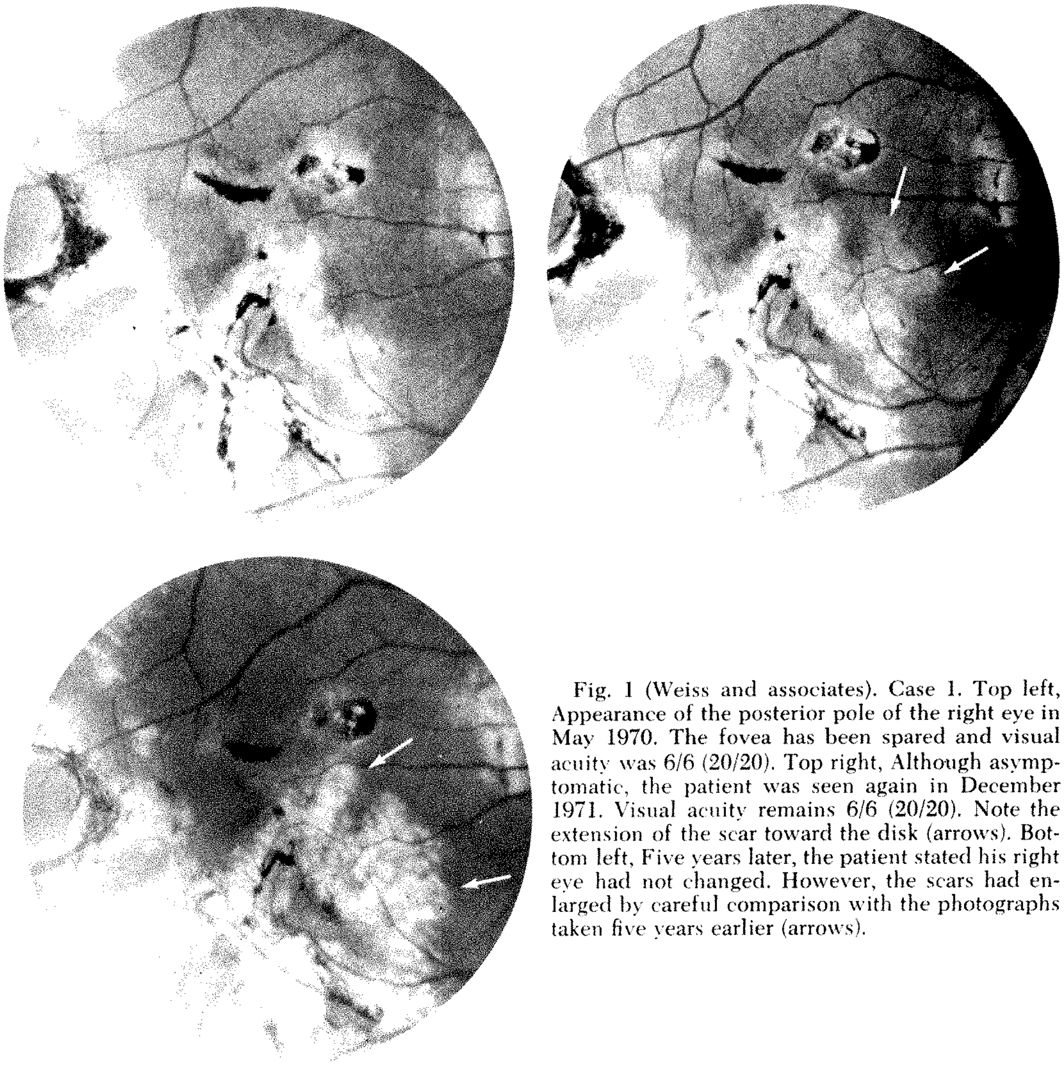


Fig. 1 (Weiss and associates). Case 1. Top left, Appearance of the posterior pole of the right eye in May 1970. The fovea has been spared and visual acuity was 6/6 (20/20). Top right, Although asymptomatic, the patient was seen again in December 1971. Visual acuity remains 6/6 (20/20). Note the extension of the scar toward the disk (arrows). Bottom left, Five years later, the patient stated his right eye had not changed. However, the scars had enlarged by careful comparison with the photographs taken five years earlier (arrows).

area of activity was often, but not always, adjacent to an old choroidal scar. Over a period of weeks, the area of new activity showed mottled pigmentation and eventually atrophy of the retinal pigment epithelium and choriocapillaris resulted. There were no instances of serious elevation of the retina over these active areas and no evidence of subretinal neovascular membranes was found.

Case 2—A healthy 22-year-old white man was first seen in April 1974, with a three-week history of blurred vision in his right eye and a one-week history of blurred vision in his left eye. His occupational history revealed exposure to various types of paint, diesel fuel, and domestic animals.

On examination April 9, 1974, best corrected visual acuity was R.E.: 6/12 (20/40) and L.E.: 6/6 (20/20). External and slit-lamp examination were normal. The fundi of both eyes showed a large area of pigment alteration below which was a broad active front of gray discoloration of the pigment epthelium (Fig. 2, top left). Similar areas were present around the disk (Fig. 2, top right). In the equatorial area of the right eye at the 12 o'clock position, there was perivenous sheathing with some nearby intraretinal hemorrhages (Fig. 2, middle left).

Three days later, the visual acuity in the right eye had deteriorated to 6/60 (20/200) while the visual acuity in the left eye had remained 6/6 (20/20). Fundus examination showed that the active front had extended down to involve the fovea of the right eye and was moving closer to the fovea of the left eye.

The medical and neurologic examinations were normal as were an extensive battery of x-ray and laboratory studies including skin test for coccidiomycosis, histoplasmosis, tuberculosis; sedimentation rate, FTA-abs, Anti-nuclear antibody, and lupus erythematosis preparation. Electroretinography and electro-oculography were normal.

Eleven days later, visual acuity in the right eye remained 6/60 (20/200) and in the left eye had dropped to 6/15 (20/50). Fundus examination showed that the active fronts had continued to progress inferiorly in both eyes across the fovea (Fig. 2, middle right). Fluorescein angiography was performed (Fig. 2, bottom).

Visual acuity in the left eye dropped to 6/30 (20/100), but one month later visual acuity had improved to 6/24 (20/80) in the right eye and 6/21 (20/70) in the left eye. The lesions had extended to the inferior arcades leaving extensive pigment alteration across the entire posterior pole.

Two months after initial examination, visual acuity had improved to R.E.: 6/6 (20/20) and L.E.: 6/21 (20/70). Four months later the visual acuity in the left eye had improved to 6/18 (20/60) and by six months had returned to 6/6 (20/20).

When re-examined 2½ years later the patient stated that there had been no change in vision since the initial course of his disease. Comparison of fundus photographs, however, showed extension of scarring along the superior arcades and around the disk in the left eye, marked pigment clumping, and several areas of subretinal fibrous proliferation.

This patient is remarkable in the symmetry of his disease, his visual recovery, and the perivascular sheathing. The visual recovery possibly can be attributed to a failure of the disease to destroy the choriocapillaries in the fovea.

Case 3—A 44-year-old white man developed a spot on his right eye in July 1970. At the time, the patient was working in a sheet metal shop and was exposed to various fumes of unknown type. Since 1958, he had performed greenhouse work and was exposed to vapone, various kinds of fertilizer, malathrone, Triban, and nicotine bombs.

When first seen on Sept. 16, 1970, his visual acuity was 6/6 (20/20) in both eyes. External and slit-lamp examinations were normal. Fundus examination revealed multiple serpiginous and isolated choroidal scars in both eyes. A large scar extending from the disk to just above the fovea in the right eye appeared with modest pigment mottling.

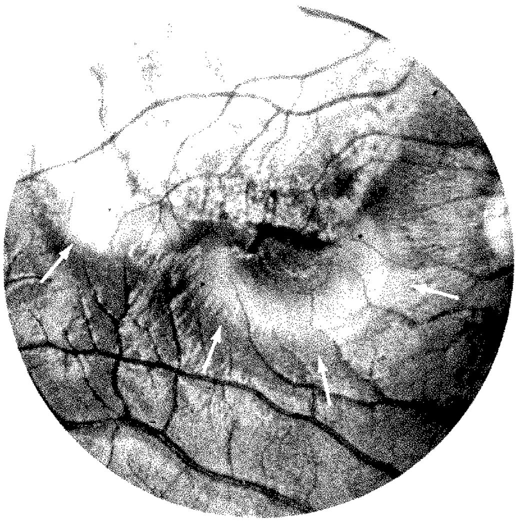
In October 1970, the patient had a systemic examination. Significant findings were a positive tuberculin skin test (second strength), a dental abscess, and mild chronic maxillary sinusitis. He was given isoniazid, 300 mg daily, and pyridoxine HCl, 50 mg daily.

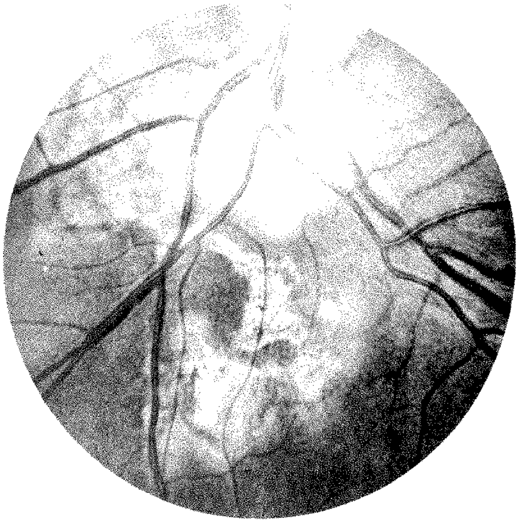
The patient was examined regularly; in July 1974 an active lesion developed in the left macular area, nasal to an older choroidal scar (Fig. 3, top left). Visual acuity in the left eye was reduced to counting fingers. No new activity in the right eye was noted, and the patient stated that there had been no change in his vision. However, on careful review of the fundus photographs, extension of the round scar below the fovea had occurred in a centripetal manner toward the disk.

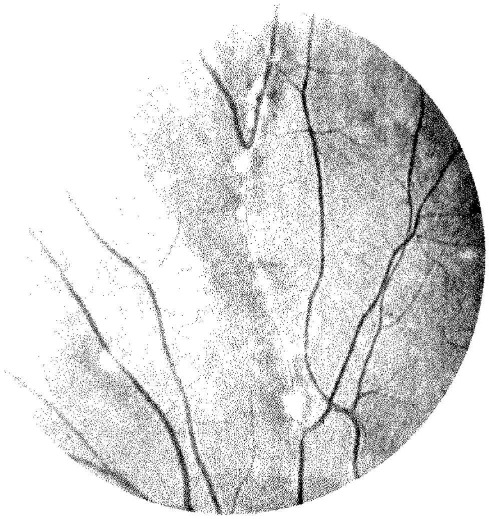
The patient's left eye was treated with a retrobulbar injection of 40 mg of methylprednisolone acetate (Depomedrol). Six weeks later, visual acuity had improved to 6/60 (20/200) in the left eye and the area of previous activity showed pigment mottling (Fig. 3, top right). Additionally, several isolated round areas of fresh activity were present along the superior arcades.

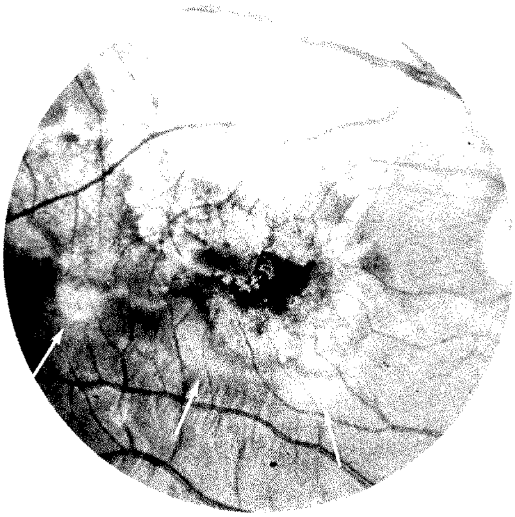
In March 1975, visual acuity in the left eye had decreased to counting fingers. Fundus examination revealed a resolving lesion with pigment mottling involving the foveal area. The right eye was unchanged. In June 1976, visual acuity was counting fingers in the left eye and 6/15 (20/50) in the right eye. In the left eye, there was further extension of the scarring, which involved a broad area of the posterior pole bounded by the vascular arcades (Fig. 3, bottom left). No new activity was present at this time. The electroretinogram and electro-oculogram were both normal. Goldmann perimetry of the right eye showed scotomas of variable depth corresponding to the fundus lesions (Fig. 3, bottom right). The patient showed asymptomatic progression, centripetal progression, and temporary visual recovery.

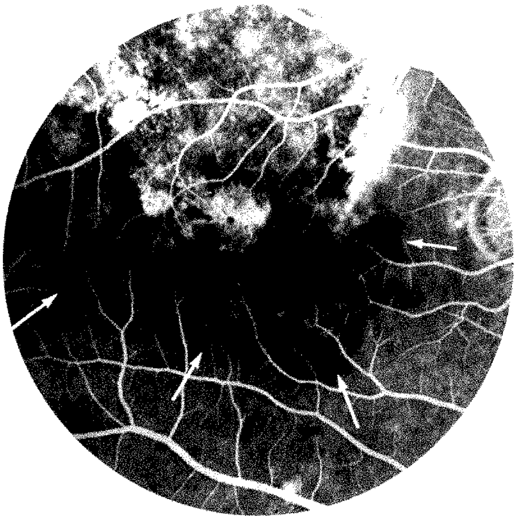
Case 4—A healthy 46-year-old white man devel-











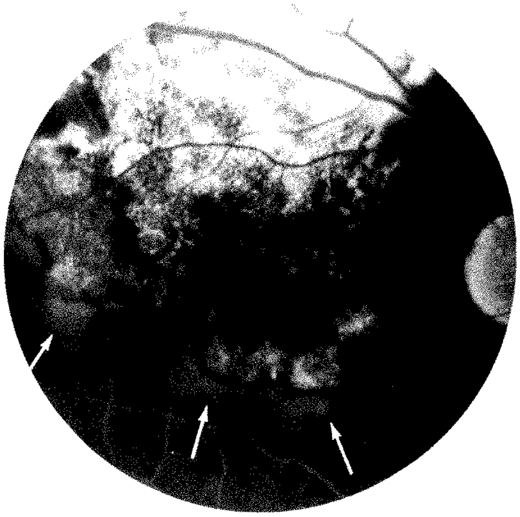


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Fig. 2 (Weiss and associates). Case 2. Top left, Posterior pole of the right eye on April 9, 1974, showing a broad active front (arrows) extending down across the posterior pole, leaving extensive pigmentary changes in its wake. Top right, Simultaneous activity was also present around both disks. Middle left, Equatorial area of the right fundus showing focal and diffuse areas of perivenous sheathing. Middle right, Eleven days later, the active front extended inferiorly (arrows). Bottom, Fluorescein angiography of the right eye in middle right. The active front is densely hypofluorescent in the early phase (arrows) (bottom left); and diffusely hyperfluorescent in the late phase (arrows) (bottom right). The older areas of involvement near the superior vascular arcades show extensive pigment changes with mottled hyperfluorescence.

oped a sudden onset of reduced visual acuity in the left eye in 1970, which continued to deteriorate until 1974. Visual acuity remained good in his right eye until January 1974, when he suddenly noted blurring of vision. The patient was a race horse trainer and was exposed to a variety of liniments as well as dimethyl-sulfoxide.

On examination Jan. 14, 1974, best-corrected visual acuity was 6/15 (20/50) in the right eye and counting fingers in the left eye. External and slit-

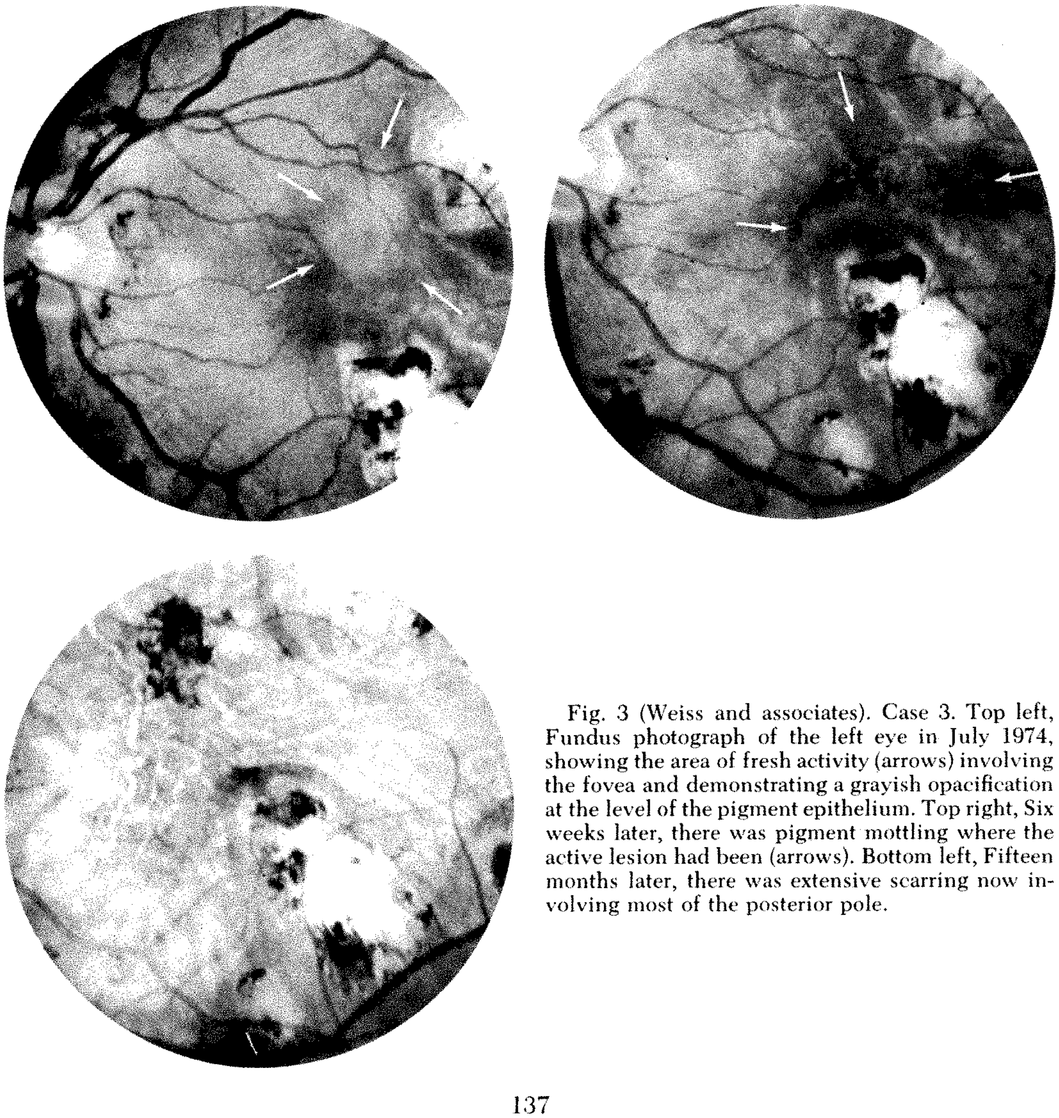


Fig. 3 (Weiss and associates). Case 3. Top left, Fundus photograph of the left eye in July 1974, showing the area of fresh activity (arrows) involving the fovea and demonstrating a grayish opacification at the level of the pigment epithelium. Top right, Six weeks later, there was pigment mottling where the active lesion had been (arrows). Bottom left, Fifteen months later, there was extensive scarring now involving most of the posterior pole.

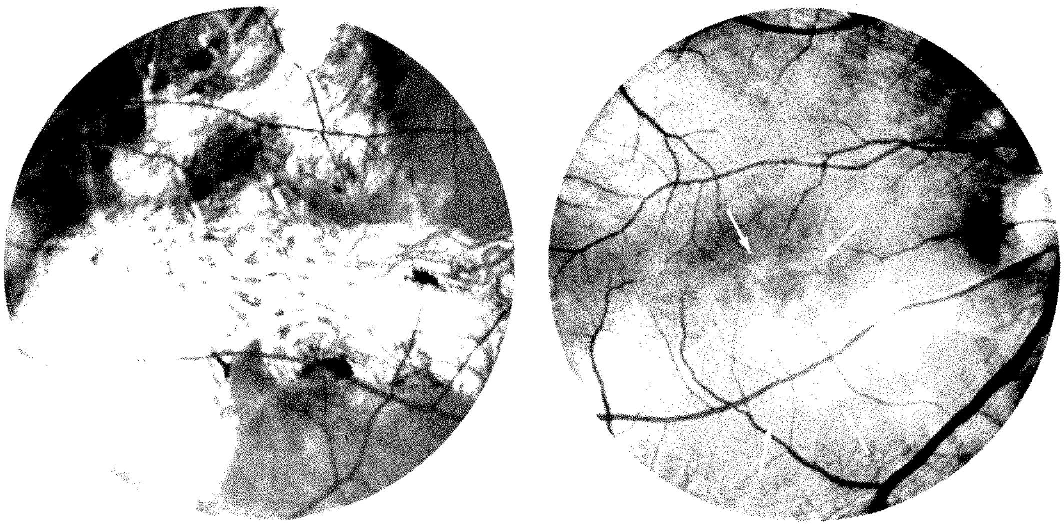
lamp examinations were normal. In the posterior pole of the left eye was a large serpiginous area of loss of the pigment epithelium, choriocapillaris, and inner choroid (Fig. 4, top left) without any new activity. In the right eye, an active area of gray opacification at the level of the pigment epithelium was present at the inferior margin of the fovea. Several small choroidal scars were present nasal to the fovea (Fig. 4, top right).

The patient was treated with oral prednisone, 40 mg daily, for six months and over this period of time visual acuity in the right eye gradually improved from 6/15 (20/50) to 6/6 (20/20).

In June 1976, the patient stated that his vision had not changed over the past two years. However, the appearance of the fundus in both eyes had changed. In the left eye, the large serpiginous scar had extended in a centrifugal manner temporally and along the superior arcades. In the right eye, additional scars were present superior to the macula and the area of new activity in 1974 showed pigment dispersion and atrophy of the inner choroid (Fig. 4, bottom). The patient showed both visual recovery following parafoveal involvement and asymptomatic progression in the right eye.

RESULTS

The clinical course of the disease in all of the patients was characterized by multiple recurrences with the development of



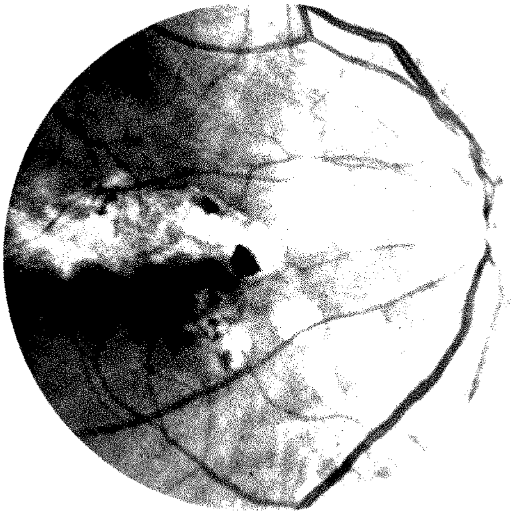


Fig. 4 (Weiss and associates). Case 4. Top, Fundus appearance of the right and left eyes in January 1974. The active area in the right eye is indicated by arrows. Bottom left, Two years later, additional scarring was present superioral to the fovea and the area of activity seen two years previously beneath the fovea demonstrated pigment changes and loss of choriocapillaris.

new scars and extension of old scars. There was often a discrepancy between the patients' histories regarding progression of their disease and the findings on fundus examination. Five of the patients were unaware that their disease had continued to progress because their recurrences were not near the macula and were, therefore, not recognized. Progression might not have been detected by the examiners if serial fundus photographs had not been available. Most of the patients had multiple and complex chorioretinal scars and new areas of activity or scarring were sometimes subtle and detectable only by close scrutiny of serial photographs. Serial fundus photography provided us with the information that the disease may progress in the absence of symptoms and that progression can continue for up to ten years, the maximum duration of our follow-up.

Most of the eyes in our series showed progression of the disease in a centrifugal manner, beginning near the disk and extending outward toward the periphery. However, in three eyes in our series, there was either centripetal progression or new activity in an area that had been previously bypassed. In one of these cases, visual acuity was permanently affected.

Visual acuity was affected only when the foveal or parafoveal areas were directly involved by the disease. In nine of the 17 eyes, visual acuity dropped to a variable degree during foveal or parafoveal involvement, but subsequently improved as the acute lesion resolved (Table 1).

In some cases, visual recovery was remarkable despite significant initial reduction in vision.

In another six of the 17 eyes in our series, the fovea or parafoveal areas were affected and visual acuity dropped but did not recover. Final visual acuities were 6/12 (20/40) (one eye); 6/15 (20/50) (one eye); 6/30 (20/100) (one eye); 6/60 (20/200) (one eye); hand movements (two eyes). In the remaining two of the 17 eyes, the foveal and parafoveal areas were not involved at all and visual acuity was unaffected.

At the conclusion of our study, no patient was legally blind and only three of the 17 eyes had visual acuity of less than 6/60 (20/200) (two had hand movements as reported above, and one eye that recovered visual acuity to 6/30 (20/100) subsequently developed foveal involvement again and became counting fingers). Final visual acuity in these 17 eyes is shown in Table 2.

Foveal or parafoveal involvement with its attendant reduction in visual acuity is common in serpiginous choroidopathy and occurred in 15 of the 17 eyes. The

TABLE 1

PATIENTS WHO DEMONSTRATED RECOVERY OF VISUAL ACUITY AFTER FOVEAL OR PARAFOVEAL INVOLVEMENT

Patient No.	Worst Visual Acuity	Best Recovered Visual Acuity	Time To Maximum Recovery (mos.)
1	6/24 (20/80)	6/6 (20/20)	4-9
2	6/60 (20/200)	6/6 (20/20)	2
3	6/30 (20/100)	6/6 (20/20)	4-6
4	6/60 (20/200)	6/9 (20/30)	12
5	6/21 (20/70)	6/9 (20/30)	3
6	6/15 (20/50)	6/6 (20/20)	6
7	Counting fingers	6/15 (20/50)	22
8	Counting fingers	6/30 (20/100)	3
9	Counting fingers	6/30 (20/100)	8

TABLE 2
FINAL VISUAL ACUITIES IN ALL 17 EYES OF THE STUDY

Visual Acuity	No. of Eyes
6/6 (20/20)	6
6/9 (20/30)	1
6/12 (20/40)	1
6/15 (20/50)	2
6/21 (20/70)	1
6/30 (20/100)	2
6/60 (20/200)	1
Counting fingers	1
Hand movements	2

reduction of visual acuity was not necessarily permanent, and in our series it was common to recover some or all central vision.

Involvement of a given area by this disease does not necessarily result in a permanent absolute scotoma, which was confirmed by our findings on quantitative perimetry and sequential Amsler grids. Goldmann perimetry was performed on six of the nine patients. Scotomas corresponding to the fundus lesions were present in all the patients tested; however, the scotomas were not absolute and usually showed a dense center with a lighter surrounding area. Two patients administered Amsler grid fields to themselves on a daily basis on eyes with recurrent activity near the fovea. Both reported on several occasions that early in the course of an acute lesion, there was an absolute scotoma, but over a period of weeks, the scotomas became less dense and vision improved in those areas.

Seven of the nine patients received corticosteroids in variable amounts and by various routes, but the course of the disease was not noticeably altered by this treatment. Electroretinography and electro-oculography were performed on eight of the nine patients, and both tests were recorded within normal limits for all eyes.

Although the characteristic appearance of serpiginous choroidopathy in its acute and chronic stages was noted in all of our patients, there were several interesting and unusual variations. Inflammatory signs, such as cells in the vitreous or aqueous, are known to be uncommon or at most, of minimal proportions in this disease. Most of our patients had a sudden onset of a "blind spot" in their vision in the absence of any pain, redness, or photophobia. One patient developed a fairly extensive vitritis with 2 to 3+ cells, and another developed a mild vitritis along with perivascular sheathing and intraretinal hemorrhages in the far periphery of one eye, which had extensive activity in the posterior pole.

The disease usually begins in the posterior pole, often adjacent to the disk, and extends centrifugally. However, in one of our patients with a classical serpiginous scar in the posterior pole of one eye, the second eye had a normal posterior pole by ophthalmoscopy and fluorescein angiography. In the nasal periphery, between the equator and pars plana, there was a small circumferential scar at one end of which a typical acute lesion was found with gray-green discoloration of the pigment epithelium, which was densely hypofluorescent early in the fluorescein angiogram and diffusely hyperfluorescent in the late phase.

One patient, again with the characteristic fundus appearance, who had experienced multiple typical recurrences over many years, developed a recurrence in which the new lesions were multiple, small, isolated round white plaques typical of those seen in acute posterior multifocal placoid pigment epitheliopathy.[5,6] These isolated lesions coalesced and left a typical serpiginous scar with loss of the retinal pigment epithelium and choroid.

Detailed medical and social histories were obtained from all patients in our series. No patient reported any systemic illness or symptoms before or during the onset of disease or during any of the acute recurrences. Two patients had known tuberculin sensitivity; two had mild hypertension; one was diabetic; one had glaucoma; and one had maxillary sinusitis.

DISCUSSION

The earliest description of serpiginous choroidopathy was mady by Junius[7] who called it parapapillary and central retinochoroiditis. Sorsby[8] described and illustrated what appeared to be several typical cases of serpiginous choroidopathy but classified them as a peripapillary type of choroidal sclerosis. Franceschetti[9] pointed out there were two groups of diseases that had similar appearance but which had different clinical courses. One group, pigmented paravenous chorioretinal degeneration, was relatively stationary and probably degenerative. Peripapillary choroidal sclerosis would presumably also fit into this group. Franceschetti's second category was called helicoid peripapillary chorioretinal degeneration, and was described as a progressive disease that represents what we now call serpiginous choroidopathy.

The clinical features and fluorescein angiographic findings in serpiginous choroidopathy have been well described by Schatz, Maumenee, and Patz,[3] Hamilton and Bird,[2] and Laatikainen and Erkkila.[1] In the inactive stage, serpiginous irregular scars are seen, which represent loss of the retinal pigment epithelium and choriocapillaris associated with varying degrees of hyperpigmentation and fibrous tissue proliferation. Since the disease often begins in the peripapillary area, the scars often interface with the disk and pursue an irregular course. Large choroidal vessels in the base of the scars frequently are the one visible remnant of the choroid. In the early fluorescein angiogram, these inactive scars are hypofluorescent because most of the choriocapillaris is absent. As the angiogram proceeds, an increasing hyperfluorescence is seen at the margins of the scar as fluorescein diffuses into the scarred area from the bordering normal choriocapillaris. In the late angiogram, the scars themselves are diffusely hyperfluorescent as the dye stains the sclera and fibrous tissue.

In the active stage, a gray or cream-colored opacification develops beneath the retina at the level of the retinal pigment epithelium. Often, the active area is adjacent to an old scar. There is no serous elevation of the overlying sensory retina and other inflammatory signs are rare. The acute lesion usually lasts several weeks, eventually becomes lighter in color, and over the subsequent months, shows pigment dispersion and takes on the appearance of the other scarred areas with atrophy of the retinal pigment epithelium and choriocapillaris, and variable fibrous tissue and pigment proliferation.

The fluorescein angiographic appearance of the acute lesion is characteristic. The active lesion is initially densely hypofluorescent and remains so until late in the angiogram when it becomes variably hyperfluorescent. This angiographic appearance could represent either blockage of underlying fluorescence because of opacification of the pigment epithelium or primary closure or nonperfusion of the choriocapillaries. We believe the resulting scar, which indicates loss of both the pigment epithelium and choriocapillaris, points to the choriocapillaris as the location of the initial pathologic process.

Chisholm, Gass, and Hutton[4] stated that the progress of serpiginous choroidopathy is usually centrifugal, beginning at the disk and extending outward. However, three eyes in our series showed centripetal progression, beginning in an extrapapillary location and progressing toward the optic disk. Patients whose fovea has been bypassed are not safe from future foveal involvement. One eye in our series lost central vision through such centripetal progression.

The possibility of visual recovery after foveal or parafoveal involvement has not been emphasized previously. Laatikainen and Erkkila[1] recorded visual recovery in six of the 18 eyes in their series. Their average duration to recovered vision was five months. Hamilton and Bird[2] reported a visual improvement from 6/60 (20/200) to 6/9 (20/30) in one eye of the five patients they described, but did not otherwise provide follow-up data on vision. Schatz, Maumenee, and Patz[3] did not find visual recovery in any of the nine patients in their series, but their follow-up was limited.

The patients in our series were examined closely and regularly, and we found that visual recovery was not uncommon. In nine eyes with foveal or parafoveal involvement in which visual acuity dropped from 6/15 (20/50) to counting fingers, variable visual recovery occurred. The most dramatic example was in our one-eyed patient in whom vision dropped to counting fingers and recovered to 6/15 (20/50). Recovery of vision generally occurred slowly over a period of months (Table 1) and was more likely to occur in eyes in which the edge of the active lesion involved the fovea. Our data support this observation.

Laatikainen and Erkkila[1] found a high incidence of active or presumed tuberculosis in their patients and treated most of them with aggressive antituberculous therapy with poor results. The patients in Hamilton and Bird's[2] series all had normal general medical evaluations. Schatz, Maumenee, and Patz[3] reported that one of their patients had influenza and a second had sinusitis, but otherwise their medical histories were unremarkable. In our series, two patients had tuberculin sensitivity without evidence of active tuberculosis; one was diabetic and one had glaucoma.

SUMMARY

We closely observed nine patients (17 eyes) with serpiginous choroidopathy for time periods ranging from two to ten years. There were seven men and two women with ages ranging from 22 to 58 years, average age was 46 years. Eight patients were bilaterally affected; one patient had only one eye. The clinical course of the disease was characterized by multiple recurrences. The recurrences and progression of the disease often were not noticed by patients if the macular area was not involved. Serial fundus photographs proved to be invaluable in establishing progression. Although the disease usually progresses in a centrifugal manner from the disk toward the periphery, three patients demonstrated centripetal progression and one of these three permanently lost central acuity. Visual acuity was affected only when the foveal or parafoveal areas became involved, but in nine of the 17 eyes, visual acuity returned to a variable degree. None of the patients had any general systemic illness. However, five of the patients reported chronic exposure to an unusual variety of chemicals.

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BACTERIONEMA MATRUCHOTII OCULAR INFECTIONS

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Bacterionema, a member of the same order as *Actinomyces* and *Nocardia*, has been isolated from the oral cavity of man and other primates but has not previously been associated with ocular disease. We report herein three cases in which *B. matruchotii* may have contributed to the pathogenesis of ocular suppuration.

MATERIAL AND METHODS

Laboratory reports of all specimens submitted to us from Jan. 1, 1973, to Dec. 31, 1977, were reviewed. *Bacterionema matruchotii* was isolated from three clinical specimens of ocular infections submitted during this five-year period. The corneal scrapings or intraocular fluid had been smeared onto glass slides for gram and acid fast stains and inoculated directly to blood agar, chocolate agar, Sabouraud agar, brain-heart infusion broth, and thiol broth or thioglycollate. Thiol broth and thioglycollate were supplemented with polysorbate, vitamin K, hemin, and rabbit serum to maintain a reduced oxygen concentration. After inoculation the broth was incubated at 35° C. When turbidity was detected, a sample was inoculated onto 5% horse blood and brain-heart infusion plates for incubation at 35° C in a candle jar (10% CO_2) and an anaerobic jar, prepared with anaerobic grade CO_2 and the addition of heavily inoculated plates of *Bacillus subtilis* to remove residual O_2. Gas chromatography was performed with the Dohrmann Anaerobic Bacteriology Analyzer using PYG medium.[1] The characteristics of the organism identified as *B. matruchotii* were similar in each of the three isolates (Table 1).

CASE REPORTS

Case 1—A 55-year-old man sustained bilateral corneal alkali burns in 1955. The left eye developed fungal endophthalmitis with subsequent phthisis and was enucleated in 1959. A Girard keratoprosthesis was inserted in the right eye after two penetrating keratoplasties failed. The postoperative visual acuity remained at 6/15 (20/50) without correction until January 1975, when he spontaneously developed a red painful eye. Examination two days later revealed visual acuity of light perception, tenderness of the globe, a purulent conjunctival discharge, and corneal suppuration. The anchoring skirt of the keratoprosthesis had resorbed, and the prosthesis extruded soon after examination. An emergency scleral graft was sutured over the central corneal defect (Fig. 1). Gram stain of intraocular fluid showed inflammatory cells but no organisms; culture showed growth only in the thioglycollate broth, from which *B. matruchotii* and *Propionibacterium avidum* were isolated (Table 2). The antibiotic sensitivity pattern is summarized in Table 3. Initial treatment was topical gentamicin (7.8 mg/ml) and bacitracin (10,000 units/ml) at two-hour intervals; subconjunctival gentamicin (20 mg) and methicillin (100 mg); and intravenous gentamicin (180 mg/day). On the second hospital day, parenteral erythromycin and vancomycin HCl and subconjunctival vancomycin HCl were added. Two days later, the patch graft dehisced, requiring excision and replacement of the scleral graft. Postoperative treatment was topical gentamicin and bacitracin, subconjunctival gentamicin and vancomycin HCl, and parenteral gentamicin and cephalothin. Oral prednisone was begun on the eighth day of treatment. Ocular inflammation progressively resolved. B-scan ultrasound showed a total retinal detachment with a nonrecordable electroretinogram, and visual acuity remained light perception.

Case 2—A 52-year-old woman with chronic herpes simplex stromal keratitis and ulceration developed a painful, red left eye in July 1975. Visual acuity was hand motions at two feet. Examination showed moderate eyelid edema, conjunctival hyperemia with a serous discharge, a small hypopyon, peripheral corneal vascularization, and a central necrotic stromal ulcer and infiltrate (Fig. 2). Smears of the corneal scrapings contained polymorphonuclear leukocytes and several gram-positive, non-acid fast, filamentous bacilli; cultures yielded *B. matruchotii* from the left cornea and conjunctiva in the thiol broth, and *Staphylococcus epidermidis*, type 4,

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TABLE 1

GROWTH CHARACTERISTICS OF *B. MATRUCHOTII**

	Aerobic	10% CO_2	Anaerobic
Growth	+	++	+
Gas	−	−	−
Motility	−	−	−
Catalase	+	+	
Oxidase	+		
Lipase	−	−	
Urease	−	−	
Phosphatase	−	−	
Nitrate reduction	+		
Starch hydrolysis	+	+	
Esculin hydrolysis	−	−	
Gelatin liquefaction	−	−	
Methyl red test	−	−	
Voges-Proskauer test	+	+	
Acid production from			
Dextrose	+	+	+
Lactose	−	−	−
Sucrose	−	+	+
Maltose	−	+	+
Mannitol	−	−	−
Manose	−	+	+
Levulose	−	+	+
Galactose	−	−	−
Salicin	−	+	+
Gas chromatography			
Propionate	L		S
Acetate	S		S
Formate	S		S
Lactate	S		L
Succinate	S		S

*L designates large amount; S, small amount. No other volatile or methylated acids were detected by gas chromatography.

from the cornea in the thiol broth (Table 2). Treatment was topical penicillin G (100,000 units/ml) and sulfacetamide (300 mg/ml) at 30-minute intervals, vidarabine ointment 3% at three-hour intervals, atropine drops, and oral acetazolamide. A repeat corneal culture after four days of treatment yielded no growth, and topical prednisolone acetate 1% was begun. Because of a conjunctival allergic response after five days of therapy, the antibiotics were changed to topical gentamicin (3 mg/ml) and chloramphenicol (5 mg/ml). A T-plano soft contact lens was applied on the seventh day to aid in reepithelialization. The central ulceration progressively healed, and the antibiotics were discontinued. Examination in 1977 revealed a clear but thin central cornea, irregular astigmatism, and visual acuity of counting fingers.

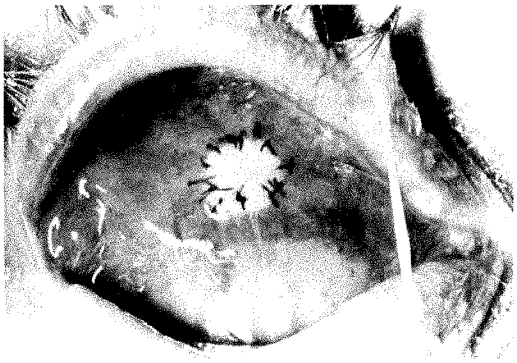

Fig. 1 (Wilhelmus, Robinson, and Jones). Case 1. The keratoprosthesis has extruded and a scleral graft is in place to maintain structure during treatment for endophthalmitis.

Case 3—A 61-year-old man underwent a penetrating keratoplasty and cataract extraction of the right eye in 1961 for a traumatic injury. He developed bullous keratopathy, which was treated with hypertonic saline. In August 1977, he was accidentally hit

TABLE 2

CULTURE ISOLATES

Case No.	Source	Smear	Thiol	Blood Agar	Chocolate Agar
1	Intraocular fluid	No organisms	*B. matruchotii* *P. avidum*	0	0
2	Cornea	Many gram + filaments	*B. matruchotii* *S. epidermidis* 4	0	0
3	Cornea	Many gram + filaments Few gram + bacilli	*B. matruchotii*	*B. matruchotii* *C. pseudodiphtheriticum* *S. epidermidis* 3	*B. matruchotii* *C. pseudodiphtheriticum* *S. epidermidis* 3

TABLE 3

ANTIBIOTIC DISK SENSITIVITIES OF *B. MATRUCHOTII**

	Case 1	Case 3
Ampicillin	S	S
Bacitracin	R	S
Cephalothin	S	S
Chloramphenicol	S	S
Clindamycin	R	S
Erythromycin	R	S
Gentamicin	S	S
Kanamycin	R	S
Methicillin	R	S
Penicillin	S	S
Polymyxin B	S	S
Neomycin	R	S
Tetracycline	S	S

S designates susceptible; R, resistant.

in the right eye with a rock and developed a red, painful eye. Examination two days later revealed visual acuity of hand motion at one foot. There was a hematoma of the upper eyelid, bulbar conjunctival hyperemia, a mucopurulent discharge, and a 7-mm corneal graft with peripheral vascularization (Fig. 3). A suppurative ulceration involved the inferior cornea with a plaque adherent to the endothelium. Gram stain of the corneal scrapings revealed inflammatory cells, several gram-positive filamentous bacilli that tended to form chains, and a few small gram-positive bacilli (Fig. 4). Culture of the corneal scrapings subsequently grew *B. matruchotii* in moderate growth on the blood and chocolate agar plates and in the thiol broth, which was sensitive to all antibiotics tested (Table 3); a few colonies of *Corynebacterium pseudodiphtheriticum* and *S. epidermidis*, type 3, were also isolated on the agar plates (Table 2). Initial therapy was intravenous gentamicin (240 mg/day) and penicillin G (12 million units/day); subconjunctival gentamicin (20 mg) and penicillin G (1 million units), twice daily;

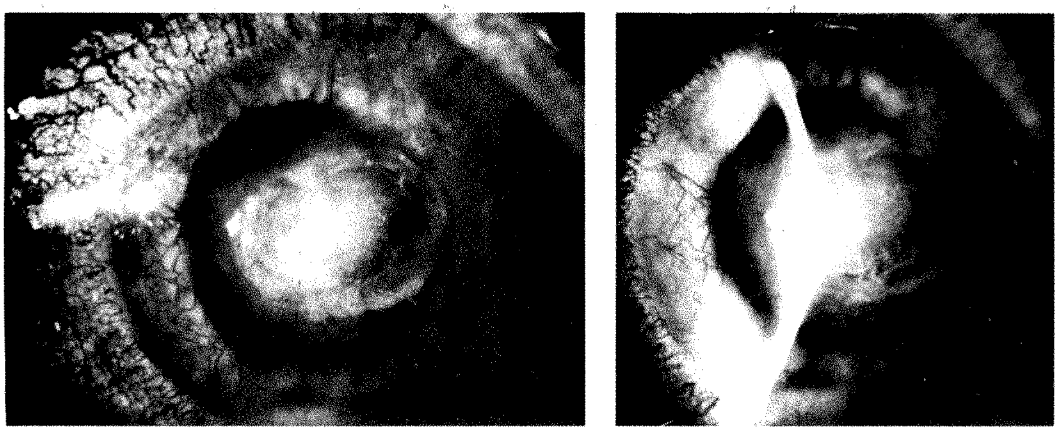

Fig. 2 (Wilhelmus, Robinson, and Jones). Case 2. A central corneal ulcer and infiltrate is surrounded by stromal vascularization. The bulbar conjunctiva is hyperemic, and a small hypopyon is present.

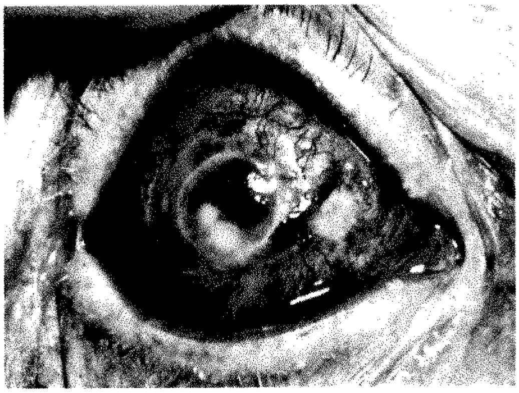

Fig. 3 (Wilhelmus, Robinson, and Jones). Case 3. A corneal ulcer is present at the inferior graft-host junction. The eye is diffusely injected and exhibits peripheral corneal vascularization.

topical gentamicin (7.8 mg/ml) and penicillin G (111,000 units/ml) at 30-minute intervals; and topical atropine 1% and phenylephrine 10%. Prednisolone acetate 1% was added on the third day of treatment. Parenteral antibiotics were discontinued after three days, and the subconjunctival injections after six days. The topical penicillin was stopped after ten days and topical gentamicin was reduced progressively over a two-week interval. The stromal infiltrate slowly resolved with re-epithelialization. Final visual acuity was limited to counting fingers by diffuse stromal scarring and edema.

DISCUSSION

Bacterionema matruchotii is a gram-positive, non-acid fast bacillus which tends to form branching filaments. The organism originally was classified as an alga and later was encompassed under the term *Leptotrichia buccalis*. In 1961 Gilmour, Howell, and Bibby[2] reclassified it under the current genus and species name in the family Actinomycetaceae. Alshamaony and associates[3] have suggested, however, that because of its propionic acid fermentation, cell wall type, DNA base composition, and certain mycolic and fatty acids, *Bacterionema* should be reclassified with the Corynebacteria.

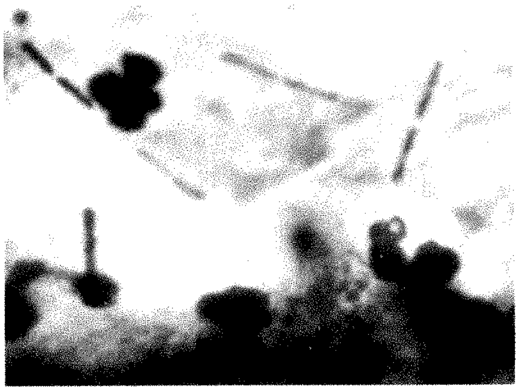

Fig. 4 (Wilhelmus, Robinson, and Jones). Case 3. Photomicrograph of the corneal scrapings showing several gram-positive filamentous bacilli surrounded by polymorphonuclear leukocytes (gram stain, ×100).

The organism has been cultivated both aerobically and anaerobically; a variety of vitamins and amino acids are required for growth which is stimulated by CO_2. The fermentation pattern, biochemical reactions, and colony types have been described.[4] Electron microscopy has shown hydroxyapatite crystals associated with cytoplasmic membranes, making it the only microorganism in which intracellular calcification has been shown in vitro.[5] Systemic pathogenicity of the organism has been tested in experimental animals, which exhibited nodular suppuration after subcutaneous and intraperitoneal injections of suspensions of pure culture isolates.[6,7] The bacterium is typically found in the oral cavity, particularly in calculus and plaque deposits on the teeth, and except for a case of maxillary abscess,[8] probably does not cause periodontal disease.[9]

Each case of ocular infection in this series occurred in an eye with a local alteration of host defenses. There was no distinguishing clinical feature among the three cases. Gram-positive filamentous bacilli were detected in direct smears of the corneal scrapings from Cases 2 and 3 (Table 2). Although *B. matruchotii* was isolated from blood and chocolate agar and thiol broth from Case 3, the exclusive yield from thiol broth in Cases 1 and 2 and the observation that the greatest amount of growth of each of the isolates occurred with candle jar incubation, suggested that reduced oxygen may be a significant factor in primary isolation.

The optimal therapy of *B. matruchotii* endophthalmitis or keratitis cannot be determined by review of these cases. In the neotype description 13 of 14 strains were sensitive to all antibiotics tested.[2] The disk-diffusion antibiograms of Cases 1 and 3 (Table 3) and the clinical response of all three cases suggested that a reasonable plan for initial therapy of suspected *B. matruchotii* ocular infection would include penicillin G or a cephalosporin antibiotic.

Because of the isolation of a mixed culture of microorganisms, the exact role of *B. matruchotii* in the pathogenesis of these three cases is uncertain. However, the presence of gram-positive bacilli in the smears of the initial corneal scrapings of Cases 2 and 3 and its subsequent isolation on culture imply that the organism played a role in the infection, either as the primary pathogen or as a participant in polymicrobial disease. Previous experience with other facultative anaerobic bacteria has shown that with careful cultivation techniques opportunistic organisms may be found to be associated with severe ocular infection.[9]

SUMMARY

One case of endophthalmitis and two cases of corneal ulceration, each occurring in a structurally altered eye, were associated with *Bacterionema matruchotii*, a gram-positive filamentous bacillus.

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LYSOZYME CONTENT OF TEARS IN PATIENTS WITH SJÖGREN'S SYNDROME AND RHEUMATOID ARTHRITIS

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Meyer[1] first showed decreased tear lysozyme concentration in keratoconjunctivitis sicca. This observation was confirmed by others.[2-6] It has been suggested that the lysozyme concentration in the tear fluid is related to the rate of tear secretion.[2,7]

We report herein the relationship between the rate of tear secretion and lysozyme content in the tear fluid of patients with keratoconjunctivitis sicca and rheumatoid arthritis, patients with rheumatoid arthritis and normal tear secretion, and patients with diminished tear secretion without systemic disease. Healthy subjects with normal tear secretion were used as controls.

SUBJECTS AND METHODS

The patient's series consisted of: Group 1, 16 patients with keratoconjunctivitis sicca and rheumatoid arthritis, aged 48 to 71 years; Group 2, 11 patients with rheumatoid arthritis and normal tear secretion, aged 45 to 60 years; and Group 3, ten patients with decreased tear secretion and without systemic disease, aged 27 to 70 years. A group of 143 healthy subjects, aged 1 month to 80 years, (that is, 286 samples of tears from both eyes) with normal tear secretion were used as controls.

Tear secretion was estimated by the Schirmer's test, performed on both eyes of all subjects. Samples of tear fluid for the lysozyme estimation were obtained by a disk of Whatman 41 filter paper 5 mm in diameter, which was inserted into the lower conjunctival sac of each eye until thoroughly wetted. For lysozyme estimation the lysozyme plate method described by Bonavida and Sapse[8] was used, with the following modifications concerning the optimal substrate concentration, time of incubation, and better contrast between lytic zone and plate background. *Micrococcus lysodeikticus* (dried cells, Sigma Chemical Company) was added to the agarose solution to make the final concentration of cells 2 mg/ml. Ten milliliters of the mixture was poured into plates and left to solidify at room temperature. These plates could be stored for one week in the cold. After application of the disks on the plate, incubation was carried out for four hours at 37°C and the zone of lysis was measured by using a precision viewer. As a standard, various concentrations of hen egg white lysozyme (HEL) were used.

RESULTS

The standard curve obtained by using various concentrations of lysozyme, which served as a reference curve for the experimental results, is shown in Figure 1. The curves obtained by using different times of incubation, when the concentration of the *M. lysodeikticus* was constant (2 mg/ml), are shown in Figure 2. The curves were obtained by using a constant time of incubation and different concentrations of *M. lysodeikticus*. The best linear relationship was obtained with the four-hour-incubation period and with a concentration of 2 mg/ml of *M. lysodeikticus*.

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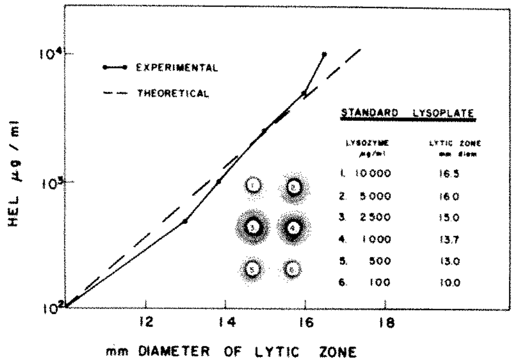

Fig. 1 (Avisar and associates). Standard curve for tear lysozyme quantitation. The diameter of the lytic zones is plotted on semilogarithmic paper.

Lysozyme content of tears of eyes with normal Schirmer's test in healthy individuals—The mean titer of lysozyme was found to be 5290 ± 2130 (SD) µg/ml of HEL, ranging from 2,500 to 10,000 µg/ml of HEL activity. Tears from each eye, analyzed separately, showed no significant difference in lysozyme content in different age groups (Table 1); therefore, the age factor was not taken into consideration. No correlation was found between the rate of tear secretion and the titer of lysozyme in tear fluid.

Lysozyme content of tears of patients with rheumatoid arthritis and normal Schirmer's test—Among the 11 patients with rheumatoid arthritis and normal tear secretion, seven had normal lysozyme content and four had a decreased lysozyme content. The mean value of lysozyme titer in tears in this group was 4509 ± 2794 µg/ml HEL activity (Table 2).

Lysozyme content in tears of patients with low Schirmer's test—Among the 16 patients with keratoconjunctivitis sicca and rheumatoid arthritis, low lysozyme content in tears was found in 12 patients and normal concentration in four patients. The mean value of lysozyme titer in tears of this group was 1136 ± 1444 µg/ml HEL activity (Table 2). Of ten patients without systemic disease, seven had normal lysozyme titer in tears, and in three the concentration was low. The mean value of lysozyme titer in tears of this group was 3645 ± 1699 µg/ml HEL activity (Table 2).

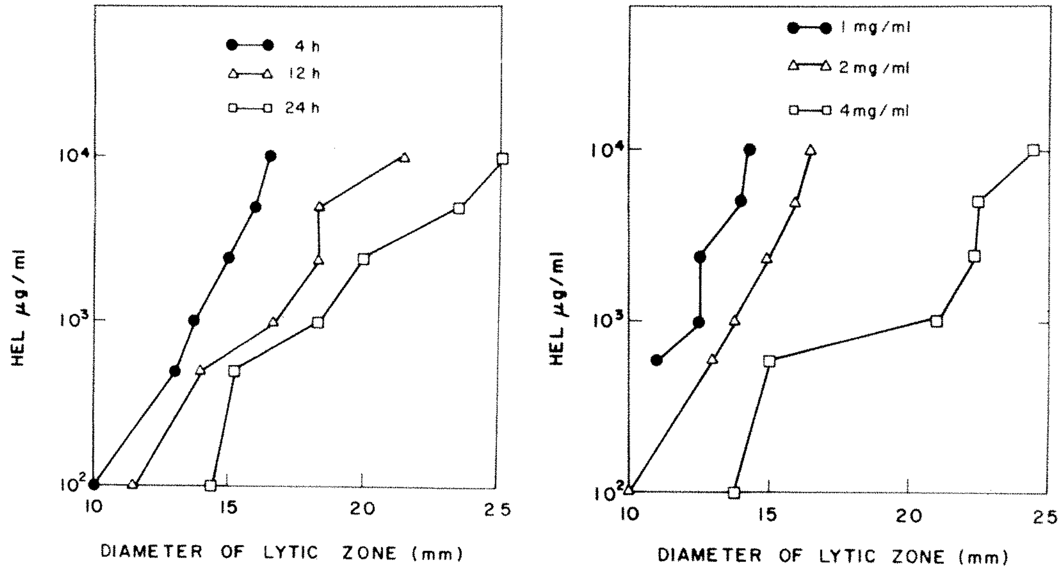

Fig. 2 (Avisar and associates). Influence of time of incubation (left) and of concentrations of *Micrococcus lysodeikticus* (right) on the diameter of the lytic zone in the lysozyme plate method of assay.

TABLE 1

RESULTS OF SCHIRMER'S TEST AND LYSOZYME CONTENT IN TEAR FLUID FROM CONTROLS, ACCORDING TO AGE GROUPS*

Age Groups (yrs)	No. of Subjects	Tear Secretion, mm/5 (min)	Lysozyme Content, μg/ml HEL
0 - 10	11	17.7 ± 4.4	5300 ± 1900
11 - 20	16	17.1 ± 3.8	5000 ± 1800
21 - 30	20	18.2 ± 2.6	4600 ± 1700
31 - 40	19	16.6 ± 4.7	5500 ± 2000
41 - 50	22	14.3 ± 2.0	5650 ± 2550
51 - 60	30	12.7 ± 2.3	5000 ± 2100
61 - 70	18	11.3 ± 2.7	5760 ± 2800
71 - 80	7	8.7 ± 0.7	5580 ± 2200
	Total 143	Mean 14.5 ± 2.9	Mean 5298 ± 2131

*The results are expressed in mean values ± standard deviation.

DISCUSSION

Our results obtained with the control group show that tear lysozyme titer is not related to the rate of tear secretion. Conflicting information exists on tear lysozyme content in patients with the keratoconjunctivitis sicca syndrome.[7,9] Few have found this enzyme decreased in keratoconjunctivitis sicca.[1-6]

Van Bijsterveld,[7] using the bacteriologic method, found the lysozyme test the most sensitive one for the diagnosis of the sicca syndrome, and under the test conditions he used, the probability of misclassification of a patient was 1%. Others[9] found a decreased titer of the enzyme in the tears of one out of six patients with keratoconjunctivitis sicca. In our experiments, the tear lysozyme titer in tears of patients with keratoconjunctivitis sicca syndrome and patients with diminished tear secretion without systemic disease, was significantly lower ($P < .001$ and $P < .05$, respectively) than that of the controls (Table 2). We assume that cases with decreased lysozyme content in tears have

TABLE 2

RESULTS OF SCHIRMER'S TEST AND LYSOZYME CONTENT IN TEAR FLUID FROM CONTROLS AND GROUPS OF PATIENTS*

Group	No. of Patients	Tear Secretion mm/5 min	P Value	Lysozyme Content μg/ml HEL	P Value
Controls	143	14.5 ± 2.9		5290 ± 2130	
Keratoconjunctivitis sicca and rheumatoid arthritis	16	2.0 ± 1.4	<.001	1136 ± 1444	<.001
Rheumatoid arthritis and normal tear secretion	11	12.8 ± 3.4	>.2	4509 ± 2794	>.2
Decreased tear secretion without systemic disease	10	3.6 ± 0.5	<.001	3645 ± 1699	<.05

*The results are expressed in mean values ± standard deviation.

a decreased production of the enzyme by the lacrimal system. This could be attributed to damage caused by specific antibodies against the striated ducts of the lacrimal gland. Such immunologic reactions in the salivary gland have been reported.[10]

The method used by us for lysozyme estimation has a number of advantages concerning sensitivity, reproducibility, and shortness. We believe it to be suitable as a screening test in various eye diseases.

SUMMARY

In 37 patients and 143 control patients we estimated tear fluid lysozyme content by the *Micrococcus lysodeikticus* agar diffusion assay. We found no correlation between the titer of lysozyme in tear fluid and the rate of tear flow. Decrease in lysozyme production was found to be a sensitive indicator of the involvement of the lacrimal system in Sjögren's syndrome.

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FAMILIAL EXUDATIVE VITREORETINOPATHY

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The syndrome of familial exudative vitreoretinopathy was originally described by Criswick and Schepens[1] in 1969 and elaborated on in 1971 by Gow and Oliver[2] who studied a large pedigree. The purposes of this paper are to suggest that the advanced stage of this disease may occur at an earlier age than had previously been noted and to describe its occurrence in a mother and daughter.

Case reports

Case 1—A woman was 7 years old when first examined for eye problems. She had been "blind in the left eye from birth." She had a "congenital, pendular-type nystagmus" and her left eye was exotropic. Vision in her right eye was 6/18 (20/60). She was the product of a full-term pregnancy and had weighed 3348.2 g at birth. No oxygen had been administered during the neonatal period.

She was reexamined recently at age 26 years. Visual acuity in her right eye was 6/120 (20/400). Intraocular pressure measured by applanation tonometry was 16 mm Hg bilaterally. She had 40 prism diopters of left exotropia, and contact B-scan ultrasonography of the left eye indicated decreased anteroposterior diameter of the globe and complete retinal detachment behind a cataractous lens. Despite the presence of a posterior subcapsular cataract in the right eye, a severe vitreous fibrosis, marked dragging of the disk, intraretinal and subretinal exudation, and a fibrovascular mass in the temporal retina could be seen (Fig. 1). A fluorescein study of that eye demonstrated an abnormal retinal capillary bed beyond the equator and massive leakage from the fibroproliferative mass in the venous phase of dye transit (Fig. 2).

Case 2—The daughter of the patient in Case 1 was examined at the age of 3 years. She was the product of a full-term pregnancy and weighed 3064.5 g at birth. No oxygen had been administered during the neonatal period. She had 45 to 50 prism diopters of exotropia, a congenital pendular nystagmus, normal corneal diameters of 11 mm bilaterally, and intraocular pressure of approximately 19 mm Hg bilaterally as determined by Schiøtz tonometry. Ophthalmoscopic examination revealed dragging of each disk (Fig. 3) and intraretinal and subretinal exudation. Vitreous changes of early degeneration and band formation were present peripherally in both eyes. Visual acuity could not be tested.

Discussion

The resemblance of familial exudative vitreoretinopathy to retrolental fibroplasia is striking, both in clinical appearance and fluoroangiographic findings. This morphologic similarity was noted by Criswick and Schepens,[1] and retrolental fibroplasia was the primary differential diagnosis they considered in their cases, although none of their six patients had been born prematurely or had been given supplemental oxygen. Furthermore, retrolental fibroplasia is apparently not a genetically transmittable disease. Gow and Oliver,[2] citing an extensive pedigree, found no cases of prematurity or oxygen administration and showed that the exudative vitreoretinopathy in their patients was inherited as an autosomal dominant characteristic. The same pedigree furnished the four cases studied with fluorescein angiography by Canny and Oliver[3] and demonstrated the common denominator of capillary bed obstruction shared by other proliferative retinopathies, particularly, retrolental fibroplasia.

This large pedigree also allowed the disease to be divided into three clinical stages, progressing from mild peripheral vitreoretinal changes in Stage I to an advanced Stage III, which included most of the findings in the left eye of our patient in Case 1. Stage I must be differentiated from mild degrees of retrolental fibroplasia, juvenile retinoschisis, heterotopic macula, and congenital retinal folds. Diseases that have elements of Stage II familial exudative vitreoretinop-

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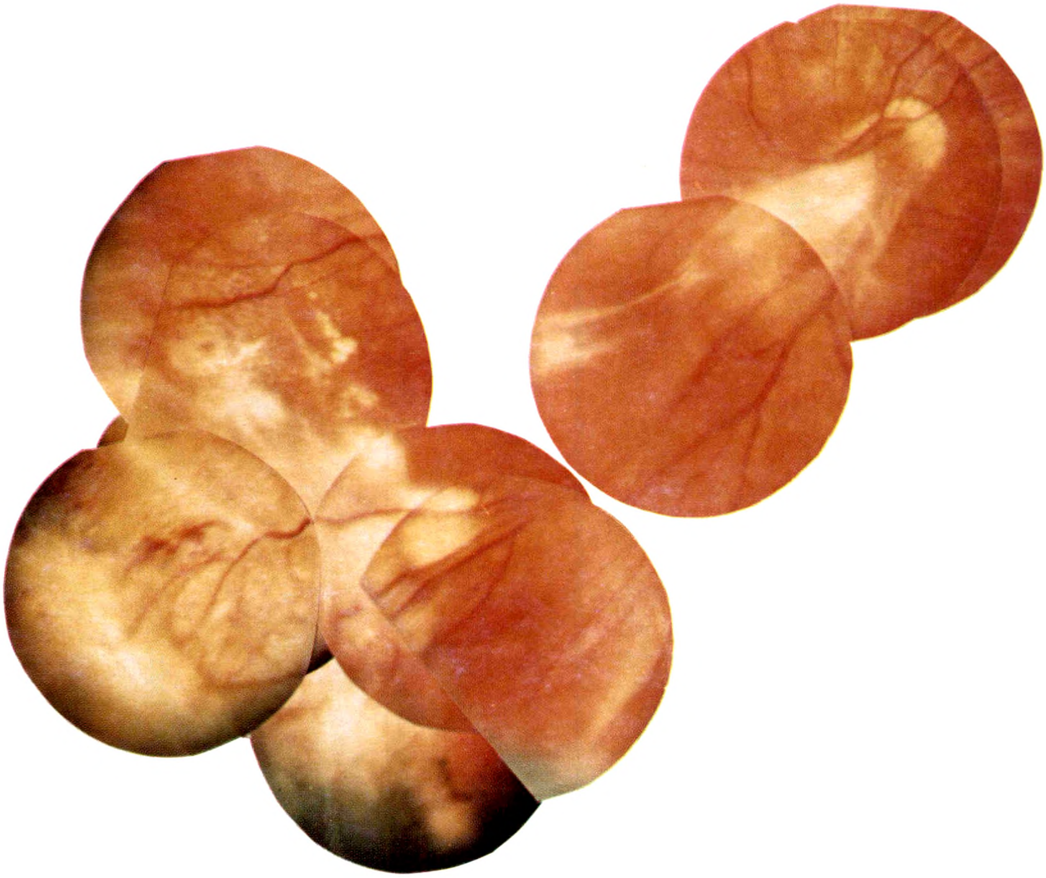

Fig. 1 (Slusher and Hutton). Case 1. Composite fundus photograph of right eye. Traction of the disk, dragging of disk vessels, subretinal and intraretinal exudation, and a temporal peripheral fibrovascular mass are visible.

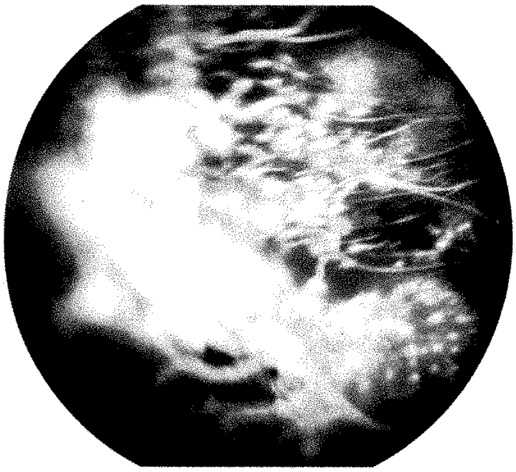

Fig. 2 (Slusher and Hutton). Case 1. Fluorescein angiogram suggesting missing capillary bed detail in equatorial area and massive fluorescein leakage from peripheral mass.

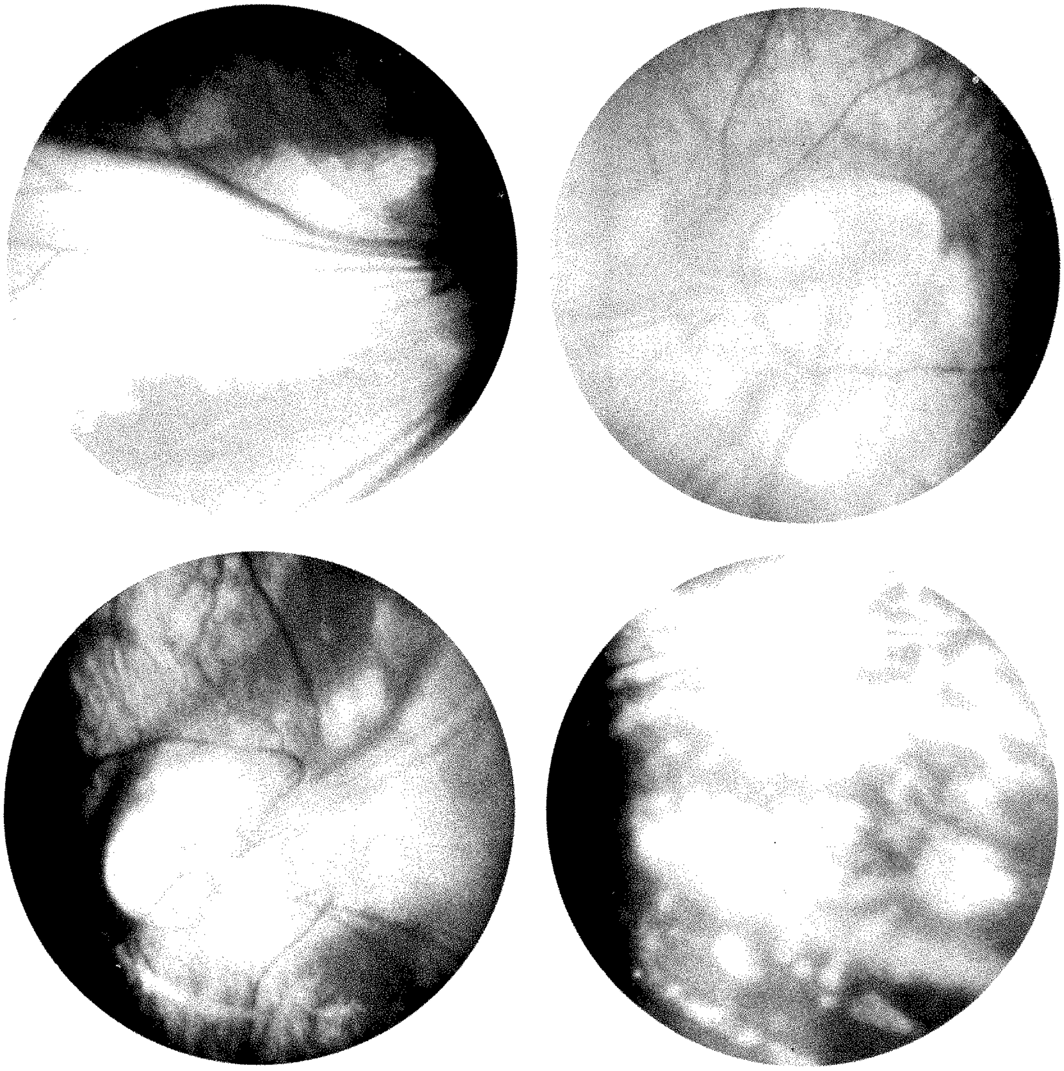

Fig. 3 (Slusher and Hutton). Case 2. Right (top) and left (bottom) fundus photographs in Case 2 showing bilateral temporal dragging of disks and exudative retinopathy.

athy are peripheral uveitis, Coats' disease, angiomatosis retinae, Eales' disease, and retinoblastoma. Although the connection has not been mentioned in other reports on familial exudative vitreoretinopathy, the same retinal exudative component and vitreous fibrosis have been present in several of our cases of *Toxocara* canis.

Both of our cases of familial exudative vitreoretinopathy prove that the syndrome's visual impairment, nystagmus, and extensive exudative retinal involvement can occur at an earlier age than had previously been thought. We believe this early involvement requires thorough examination of all members of an involved family because prompt retinal cryotherapy may help prevent the total retinal detachment and blindness that occurred in the patient in Case 1.

SUMMARY

At age 26 years a woman who had been blind in the left eye from birth had visual acuity of R.E.: 6/18 (20/60); L.E.: 6/120 (20/400), with 40 prism diopters of left exotropia. The left eye showed a decreased anteroposterior diameter of the globe and a complete retinal detachment behind a cataractous lens. The right eye had a posterior subcapsular lens opacity, severe vitreous fibrosis, dragging of the optic disk, and intraretinal and subretinal exudation with a fibrovascular mass in the temporal retina. The patient's 3-year-old daughter had 45 to 50 prism diopters of exotropia, a pendular nystagmus with intraretinal and subretinal dragging of each disk, and early degeneration and band formation in the periphery of each fundus.

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MULTIFOCAL RETINOPATHY IN BORNA DISEASE VIRUS INFECTED RABBITS

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In recent studies, inflammatory diseases of the retinal pigment epithelium as well as the bordering neuroepithelia, such as acute pigment epitheliitis,[1] acute multifocal pigment epitheliopathy,[2] and acute macular retinopathy[3] have been suggested as being responsible for multifocal alterations at the posterior pole of the eye. The cause of these spotlike chorioretinopathies has not been established. Clinical observations[3] point to an infectious process, and virus diseases or immune pathologic events have been considered as initiating factors.[4,5]

In the course of our studies on the pathogenesis of Borna disease in rabbits we became aware of a highly reproducible retinopathy. Borna disease, a slow virus infection,[6] naturally occurs in horses and sheep, and causes an encephalomyelitis with perivascular lymphocytic infiltrations and intranuclear ganglionic inclusion bodies in the brain.[7,8]

We describe herein the ophthalmologic manifestations of Borna disease in experimentally infected rabbits. Evidence indicates that comparable pathogenetic events occur under similar pathologic conditions in the human eye.

MATERIAL AND METHODS

Sixty rabbits between the ages of six and 12 weeks as well as four newborn rabbits were inoculated intracerebrally with 0.4 ml (0.1 ml) of a 10% brain suspension in phosphate-buffered saline solution. The suspension was derived from Borna disease virus (strain V[9]) infected rabbits or from a naturally infected horse. Ten rabbits received noninfected rabbit brain suspension by the same route. Five rabbits were inoculated with homogenates of persistently infected cell cultures,[9] and three rabbits with corresponding uninfected tissue culture material. Two noninfected rabbits and the mother of the four infected newborn rabbits were cohabitated with diseased animals for five months.

The anterior components of the eye were observed under focal and retrograde illumination. After application of a mydriatic solution (four drops of tropicamide and two drops of phenylephrine 10%) the fundus was examined by indirect monocular or binocular as well as direct ophthalmoscopy before infection and again on days 10 and 20 post infection, and thereafter at one- or two-day intervals.

Fundus photographs were made and fluorescein angiograms were made after injection of 1 ml of a 10% sodium fluorescein solution into the outer ear vein. A complete series of fluorescein angiograms was not possible. A total of 40 animals were photographed.

In one experimental series the animals were killed on days 1, 3, 5, 9, 13, 16, 20, 23, 28, and 34 post infection; in a second series on days 7, 15, 22, and 30 post infection; and in all other experiments one to two days before expected death. All animals were killed by injection of 3 ml of sodium pentobarbital solution into the ear vein. After removal, the eye was sectioned equatorially and disks 1.4 mm in diameter and with an average weight of 5 mg ± 0.5 mg were punched out of the retina and fixed in 2.5% glutaraldehyde in

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0.1M cacodylate buffer at pH 7.2 and 4°C overnight for histologic investigation.

Serum, cerebrospinal fluid, and aqueous humor from the various animals were tested for virus-specific antibodies by indirect fluorescence antibody tests on fixed, infected tissue culture cells as well as by complement fixation, immunodiffusion, and immunoelctrophoretic tests.[10] Retinal and choroidal disks were assayed for the presence of antigen in an indirect fluorescence antibody test by using cerebrospinal fluid from horses that had been naturally infected with Borna disease virus.[11] Retinal disks were ruptured by forcing through a tuberculin injection needle and used for infectivity assays performed by intracerebral injection of rabbits.[9]

RESULTS

No structural evidence of pathologic alteration was found in the outer eye. Of the infected animals, 30%, particularly those with severe paralysis and other clinical symptoms, displayed a delayed direct light reaction of the pupil. The remainder exhibited normal pupillary reactions until they were killed before the final stage of the disease. Examination of the fundus of the mock infected animals as well as of two rabbits held in close contact with infected animals showed no significant alteration. In all animals, infected intracerebrally and by contact, spotlike chorioretinopathic alterations were observed beginning on days 20 and 27 post infection (mean 24.1 days; S.D. 2.9 days). The isolated foci that first appeared in the anterior lower quadrant close to the equator were circular and appeared gray red; their size was 1/6 to 1/10 of the total papillary diameter. These spots always occurred alone and were of uniform size and appearance (Fig. 1). One day after appearance the foci were larger, more conspicuous, and slightly raised. The rate of enlargement of the foci increased in the next two days. Similar foci then appeared in the posterior lower quadrant and the foci enlarged to confluency. Extensive areas of the fundus were covered, and maximum affliction occurred in the equatorial zones of the lower anterior quadrant.

The process in the retina progressed with the appearance of new solitary foci that became enlarged and confluent, moving concentrically towards the optic disk (Fig. 2). By 27 to 37 days post infection all of the quadrants were afflicted in the equatorial region and at the posterior pole. Simultaneously, a coarsely grained pigmentation was visible in the anterior lower quadrant and the normal coloration of the fundus disappeared. Between days 29 and 40 post infection (mean 35.6 days, S.D. 3.6 days), a fine diffuse black granulation was visible over the complete fundus.

Fluorescein angiography of small solitary foci showed marked hyperfluorescence in early filling phases. Affliction of the pigment epithelium predominantly involved the center of each focal area. With further progression of the retinopathy, marked leakage of dye was found in later filling phases. In later stages of the disease a widespread atrophy of the choriocapillaris and loss of pigment epithelium was noted.

In two of four newborn, infected at day 1, the retinopathy progressed stepwise at intervals of approximately one month, leading to clinical neurologic symptoms only in the final stage, two months post infection. In four animals only a retinal manifestation without other clinical symptoms was detected. The animals have survived an observation period of one year.

No pathologic aberrations were observed in focus-free regions of the retina in early clinical phases (days 20 to 27)

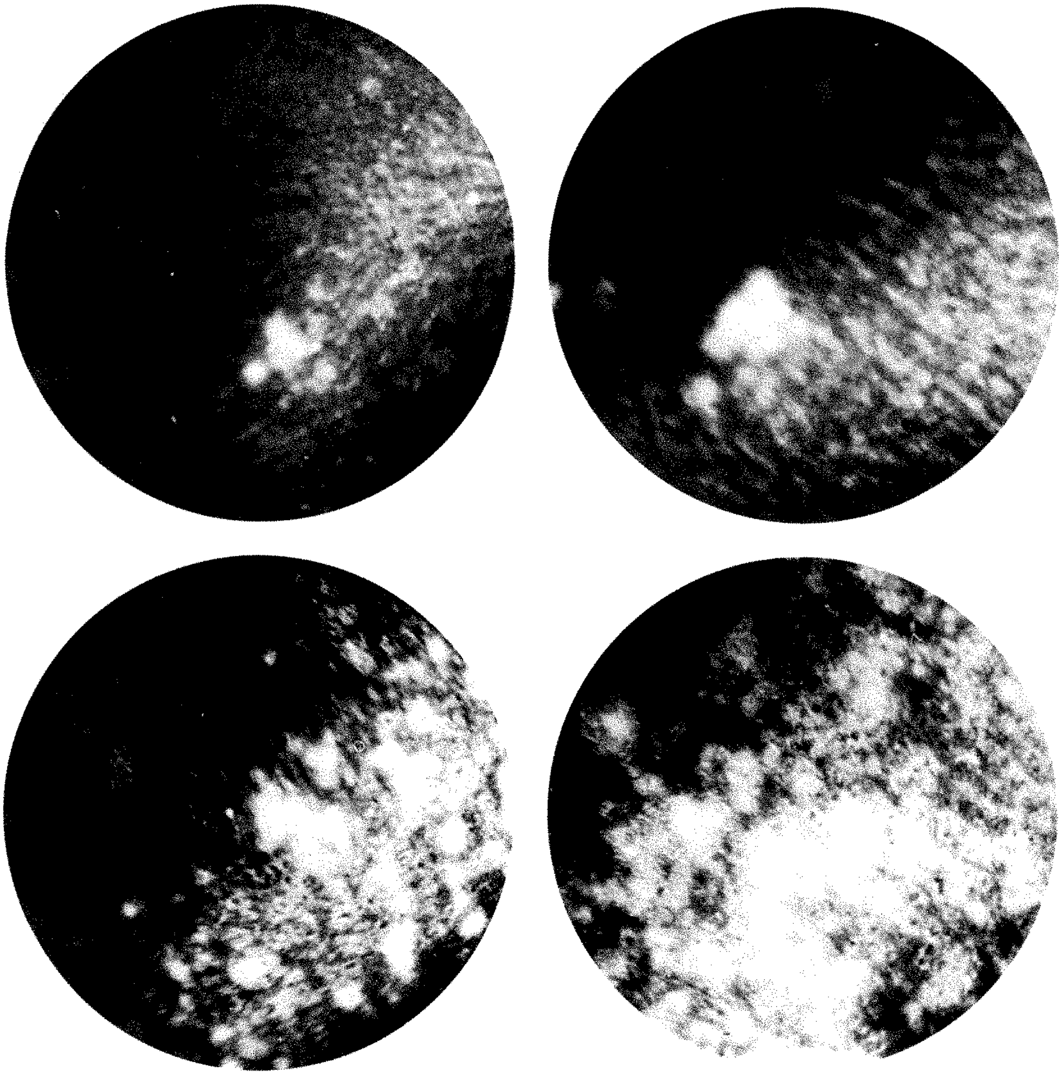

Fig. 1 (Krey, Ludwig, and Boschek): Appearance of fundus lesions in different stages of the retinopathy as shown in fluorescein angiography. The ophthalmoscopic manifestation is characterized by the appearance of equally sized spots (top) that become confluent (bottom) with progression of the disease.

(Fig. 3). Characteristic alterations were observed in preparations from focus-bearing zones of the anterior lower quadrant. A diffuse lymphocytic infiltration of the inner layers of the choroid was accompanied by an accumulation of epithelioid and plasma cells near small branches of the blood vessels. The choriocapillaris and Bruch's membrane were unchanged, whereas the retinal pigment epithelium showed signs of localized thickening and reduced pigmentation in the center of the focus. Alterations in the outer segments of the photoreceptors were coarsely granular at points, directly juxtaposed to the infiltration centers in the choroid. As the process in the retina progressed to the period in which large confluent

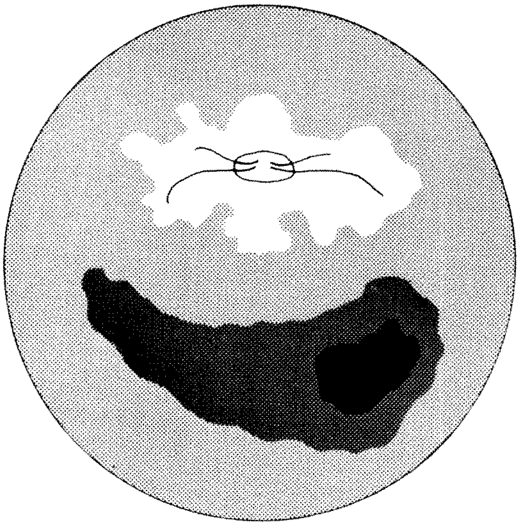

Fig. 2 (Krey, Ludwig, and Boschek): Schema of the fundus regions (right eye) where lesions are located during the progress of the disease. The black area represents the fundus zone where lesions initially appear; gray and light gray areas represent the fundus zones where additional lesions appear during the progress of the retinopathy. The vicinity of the optic disk (white area) is involved at the final stage.

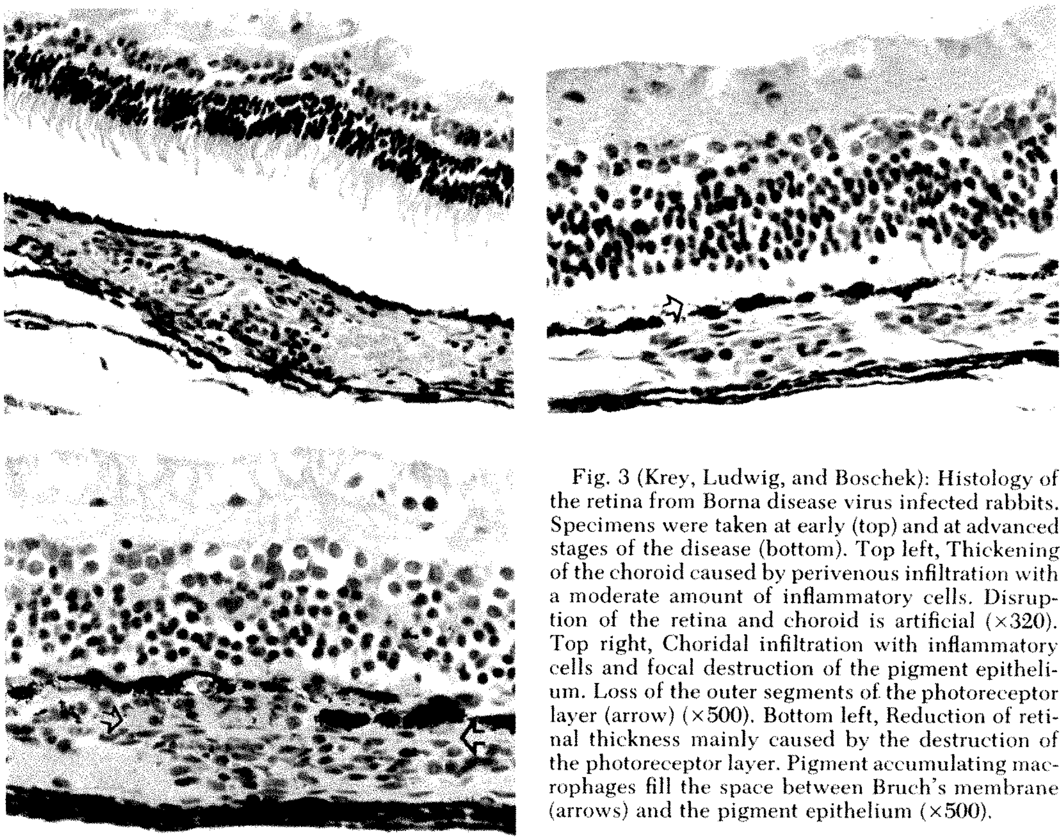

Fig. 3 (Krey, Ludwig, and Boschek): Histology of the retina from Borna disease virus infected rabbits. Specimens were taken at early (top) and at advanced stages of the disease (bottom). Top left, Thickening of the choroid caused by perivenous infiltration with a moderate amount of inflammatory cells. Disruption of the retina and choroid is artificial (×320). Top right, Choridal infiltration with inflammatory cells and focal destruction of the pigment epithelium. Loss of the outer segments of the photoreceptor layer (arrow) (×500). Bottom left, Reduction of retinal thickness mainly caused by the destruction of the photoreceptor layer. Pigment accumulating macrophages fill the space between Bruch's membrane (arrows) and the pigment epithelium (×500).

focus areas were clinically detectable, the number of epithelioid cells increased, and a general thickening of the choroid was accompanied by breaks in Bruch's membrane and destruction of the choriocapillaris.

In some areas, pigment-accumulating macrophages as well as fibrocytes filled the space between the retinal epithelium and Bruch's membrane and new branches extended from the choroidal vessels. The photoreceptors were totally missing and a general vacuolar disintegration of the plexiform layers was evident.

In the late stages the choroid was considerably thickened because of inflammatory infiltrations. The vascular system was diminished, and the choriocapillaris was missing.

Bruch's membrane was still distinguishable; however, the elastic lamina was separated from the fibrous lamina and the lumen thus formed was filled with pigment-accumulating macrophages, cell detritus, and remnants of pigment epithelium. The retina was reduced in thickness because the outer segments of the photoreceptors were missing. In some regions it was infiltrated by large pigment-bearing cells. There were no signs of any infiltration of the retina by inflammatory cells.

Indirect immunofluorescence tests of cryostat sections first showed intranuclear antigen on day 16 post infection in the ganglion layer and at day 20 post infection in the inner and outer nuclear layer and the pigment epithelium. The fluorescing particles were larger in the pigment epithelium than in all other layers and every cell carried antigen aggregations (Fig. 4). The antigen in the area of the chorioretinal inflammatory foci was identical in quantity and distribution to the adjacent focus-free areas. No specific fluorescence was seen in the photoreceptors or in the choroid. Virus-specific antigen-bearing retinal preparations that were stained with cerebrospinal fluid of uninfected horses never gave a positive reaction.

The infectivity assays showed that the retinas of animals with clinically detectable retinopathies were infectious. Each of 20 test animals injected with homogenates of 20 different retina-preparations acquired typical symptoms of Borna disease, and virus-specific antigen was found in the brains and retinas of those test animals as well. In two groups of rabbits that had been inoculated with two different infectious brain suspensions as early as 15 (16) days post infection, infectious virus was found in the retina. This was approximately one week before fundus foci could be expected by ophthalmoscopy. To determine whether infectious virus was spread throughout the retina, identical quantities of tissue were tested from the afflicted, lower anterior quadrant and from the focus-free upper posterior quadrant of the same retina. Both regions carried infectious virus. Antigen was found in both afflicted and unafflicted regions by immunodiffusion, complement fixation, and immunoelectrophoretic experiments (data not shown).

All of the 25 rabbits tested in this investigation had antibodies detectable in the serum, in the aqueous humor, and locally produced antibodies in the cerebrospinal fluid, as reported previously.[10]

DISCUSSION

Experimental Borna disease of the rabbit is always accompanied by severe retinal alterations. Outstanding features are ophthalmoscopically detectable isolated foci, which become confluent and spread over the total retina. Retinal tissue harbors Borna disease virus and specific antigen.

The results obtained in this report allowed a suggested explanation of the clin-

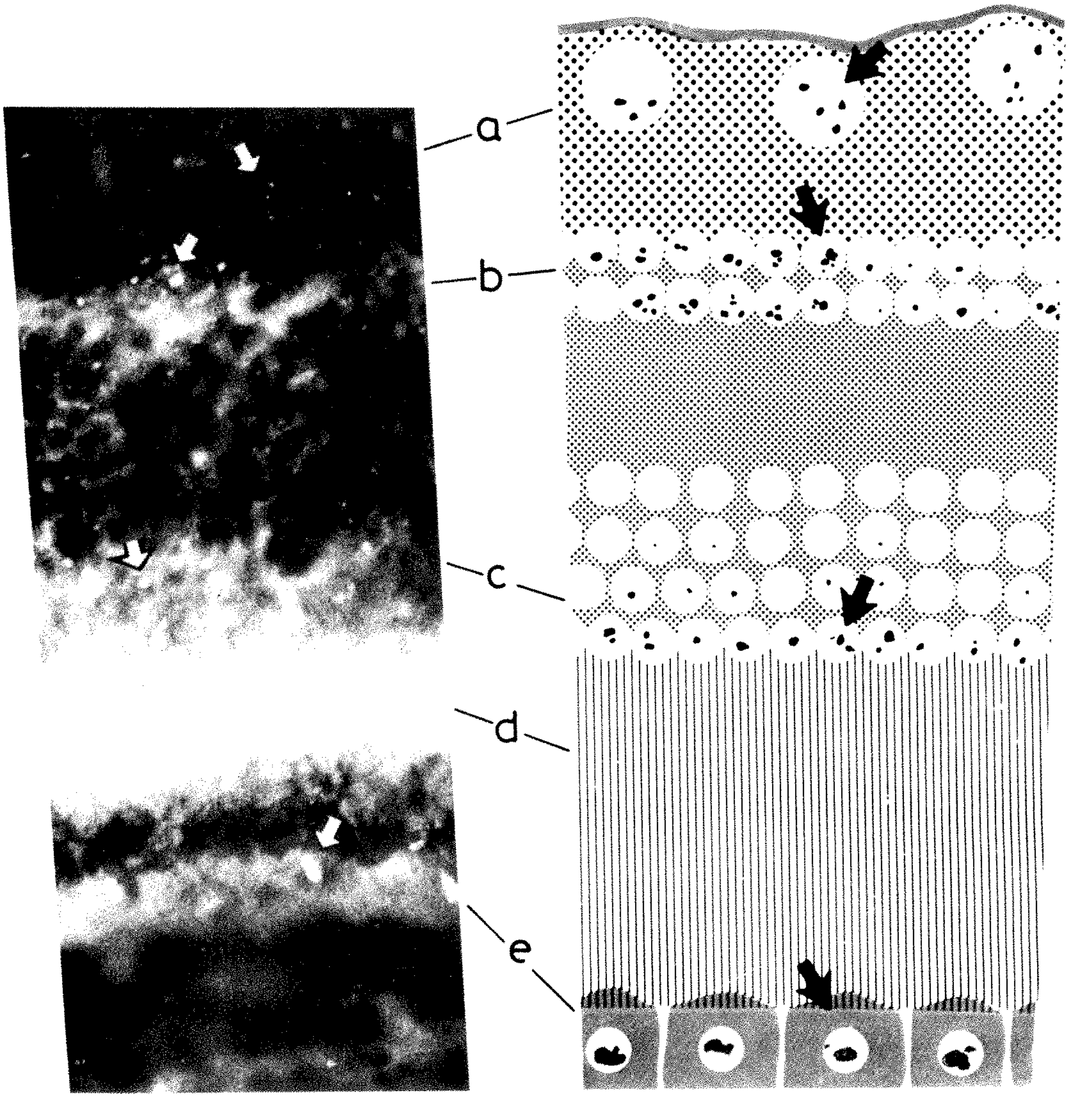

Fig. 4 (Krey, Ludwig, and Boschek): Borna disease virus-specific antigen distribution in the rabbit retina. Tissue was taken at an advanced stage of the disease from a focus-free region of the posterior pole of the eye. Left, Immunohistologic picture of a cryostat section. Right, Schema of intranuclear antigen. Antigen deposits were noted in the nuclei of the ganglion cells (a), of the inner nuclear layer (b), of the outer nuclear layer (c) and the pigment epithelium (e). The fluorescence in the photoreceptors (d) was caused by autofluorescence as revealed from preparations of uninfected retinas.

ical and histopathologic appearance. Virus and antigen were found in the unaltered retina as well as in the typical plaque-like foci of the fundus. This may indicate that the damage is not just caused by virus replication followed by cell destruction, but that an immunopathologic process gives rise to the typical alterations. This is supported by the indication that the focal destruction of mainly the pigment epithelium and the photoreceptors always occurs at positions where perivenous infiltrations have been observed in the choroid.

As drawn from our histopathologic, immunohistologic, and fluorescein-angiographic findings, the pigment epithelium probably suffers the initial damage. The focal nature of the disease is surprising because infectious virus and specific antigen did not show any focal accumulation in the retinal tissues. According to experimental findings of Waksman,[13] we speculate that small choroidal veins play an important part in the determination of the multifocal nature of the disease.

According to Correia[12] venules of the rabbit choroid originate in the center of star-shaped capillary areas facing the pigment epithelium. Small venules of this type have been shown experimentally[13] to be predilection sites of immunoreactions. In experimental Borna virus disease, most likely an accumulation of Borna disease virus specific antibodies or immunocompetent cells occurs here. The known permeability of these sinusoid venules[13,14] offers the explanation of an immune reaction in the antigen loaded juxtaposed pigment epithelium which is followed by cell destruction in this particular area. This correlates well with the hyperfluorescence of the fundus lesions in fluorescein angiography and the flatness of the individual foci in early stages of the disease. The focal inflammatory reaction of the choroid seems to be secondary to an immunopathologic process in the pigment epithelium. The prevalence of focal alterations in the anterior lower quadrant, however, cannot be explained on a vascular basis (G. L. Ruskell, Ph.D., personal communication, Feb. 2, 1978).

This phenomenon may be related to the topography of neural elements in the rabbit retina.[15] The region primarily involved during the disease correlates to a certain extent with the visual streak, a fovealike zone containing a high density of ganglion cells.

The pathway of the virus is not known but it seems to reach the retina by way of the ganglion cells via a centrifugal spreading into this area. An accumulation of antigen may first reach the necessary threshold here to initiate the immunopathologic destruction.

The focal nature of chorioretinal diseases and their preferential location in the posterior pole of the eye has also been documented in human diseases, the cause of which has pointed to virus infections such as subacute sclerosing panencephalitis, rubella, or cytomegalovirus retinopathy.[16] Therefore, there seems to be a general correlation of neurotropic virus infections and ophthalmologic manifestations in the human eye. Based on the lobular structure of the choriocapillaris in the human eye[17] an analog pathogenetic process for multifocal retinopathies might be deduced from our findings in Borna disease virus infected rabbits.

SUMMARY

Experimental infection of rabbits with Borna disease virus led in all cases to a multifocal retinopathy that paralleled the clinical neurologic symptoms. The retinal changes always became evident first in the lower anterior quadrant of the eye. Infectious virus and antigen were detected in altered and unaltered regions of the retina. Individual chorioretinal lesions showed destruction of the pigment epithelium and the photoreceptors and perivascular inflammation close to small choroidal veins. Because of maximal antigen accumulation and the focal destruction of the retinal pigment epithelium we consider this cell layer to be the initially damaged structure.

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LACK OF BLOOD GROUP ANTIGEN A ON HUMAN CORNEAL ENDOTHELIUM

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The homografted human cornea can elicit an immune response and can be destroyed by that response. Which antigenic determinants play a role in the stimulation of this immune response is unknown. Nelken and associates[1] reported that preparations of whole, minced human corneas possess A and B blood group antigens. Herold[2] found these antigens only in epithelium of 12 autopsied eyes; but questions arose as to whether his technique might have missed detection of the antigens, perhaps in low concentration, on the endothelium. Conflicting reports of the importance of ABO compatibility to keratoplasty outcome have been published.[3-7] We describe herein our search for an easily detectable blood group antigen, A-antigen, on human corneal epithelium and endothelium optimally prepared for preservation of the antigen.

SUBJECTS AND METHODS

Tissue from 15 patients undergoing penetrating keratoplasty at the Massachusetts Eye and Ear Infirmary was used for this study. Ten of the patients had keratoconusc, three had corneal edema, and two had corneal scars from trauma. All patients were blood typed by the standard agglutination techniques.[8] Five of the patients were A positive, two were A negative, one was AB positive, and seven were O positive.

Specimens of epithelium and of endothelium from the 15 human corneal buttons removed at penetrating keratoplasty were placed on glass slides coated with silicone. The specimens were obtained and processed in the operating room immediately after removal of the corneal tissue from the patient. Eight were stored at −70°C and analyzed within ten days; seven were processed and examined immediately.

To insure adherence of cells to the slides, all tissue slides were briefly heat-fixed immediately before specimen analysis. Preliminary experiments with slides of type A human red blood cells showed that A-antigen was easily detectable with the fluorescent antibody technique and with the mixed agglutination technique after heat-fixation, even if the specimen has been stored at −70°C for three months before heat-fixation.

The IgG fraction of human anti-A antiserum was prepared from normal human volunteers (blood type O positive) immunized with purified human A substance. The antiserum was found, on screening with panels of test red blood cells, to contain no detectable antibodies to rhesus antigens or to antigens M, N, Lewis, Duffy, Kidd, and Kell. The antiserum was conjugated with fluorescein isothiocyanate. This antiserum was used both for the fluorescent antibody and for the mixed agglutination experiments. The protein content of the final preparation in 0.02M Tris-citrate buffer (pH 7.5) was 3.8 mg/ml and the fluorescein-protein ratio was 7.2 μg of fluorescein per milligram of

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protein. This ratio is higher than that used by most workers; but the antiserum did not stain non-type A red blood cells nonspecifically, and therefore was considered appropriate for the experiments. The highest dilution of this antiserum capable of promoting agglutination of type A human red blood cells was 1:8,192. The highest dilution capable of producing fluorescence of human type A red blood cells was 1:32.

A control antiserum was prepared from the IgG fraction of human gamma globulin from a blood type O positive donor. The protein content of this antiserum was 1.8 mg/ml and the fluorescein-protein ratio was 5.7 μg of fluorescein per milligram of protein. Specificity of the anti-A antiserum was confirmed by human type A red blood cell agglutination reactions, and by fluorescence microscopy of type A red blood cell smears stained with the antiserum. Red blood cells to be used as the test indicator cells in the mixed agglutination experiments were obtained from blood type A, B, and O donors and prepared in 0.5% phosphate-buffered saline suspensions.

Corneal epithelial and endothelial specimens from six of the patients were evaluated by the fluorescent antibody technique in masked fashion in duplicate. Undiluted antiserum was layered onto the specimen slides and allowed to remain at room temperature for 30 minutes. The slides were then washed twice in phosphate-buffered saline solution and were examined under a Zeiss fluorescence microscope fitted with a mercury burner, UG2 and BG12 excitation filters, and appropriate barrier filters. The degree of cell membrane fluorescence was graded on a scale of 0 to 4+ and was documented photographically.

Tissue from the other nine patients was evaluated by the mixed agglutination technique.[9] Six slides of corneal epithelial smears and six slides of corneal endothelial smears were used for each of these nine patients. Anti-A antiserum was applied to three of the epithelial and three of the endothelial slides of each patient; control antiserum was applied to the remaining three slides of each tissue. After incubation at room temperature for one hour, the slides were washed four times with phosphate-buffered saline solution. Human indicator test red blood cell suspensions (0.5% in phosphate-buffered saline solution) were then layered onto the slides, which were incubated at room temperature for one hour.[10] Table 1 is an example of the tabular flow and data form used for the mixed agglutination studies of each specimen. In this example, epithelial specimens from patient 12, a blood type A patient, exhibited a positive mixed agglutination reaction with type A red blood cells when anti-A antiserum was used, but not with other type red blood cells and not when control serum was used. At the end of the incubation period, the slides were allowed to drain for one hour to allow excess red blood cells to fall from the slides as recommended by Högman.[11] The slides were covered with a cover slip secured with a drop of phosphate-buffered glycerol solution. Examination in masked fashion by phase contrast microscopy with a Leitz microscope equipped with phase optics was performed. Adsorption of red blood cells

TABLE 1

MIXED AGGLUTINATION REACTION, HUMAN CORNEAL EPITHELIAL CELLS AND HUMAN RED BLOOD CELLS

Slide*	Antiserum	Indicator Cells	Reading
Epithelium	Anti-A	A	4+ rosetting
Epithelium	Anti-A	B	Negative
Epithelium	Anti-A	O	Negative
Epithelium	Control	A	Negative
Epithelium	Control	B	Negative
Epithelium	Control	O	Negative

*Specimens from patient No. 12.

onto the corneal epithelial and endothelial cells was evaluated. The criterion for rosetting was three or more red blood cells clustered against the epithelial or endothelial cell membrane. The results were documented photographically.

RESULTS

Fluorescent antibody technique—A-antigen could not be found on the endothelium of the corneas from blood type A or AB patients (Fig. 1). Corneal epithelial cell membranes from these patients

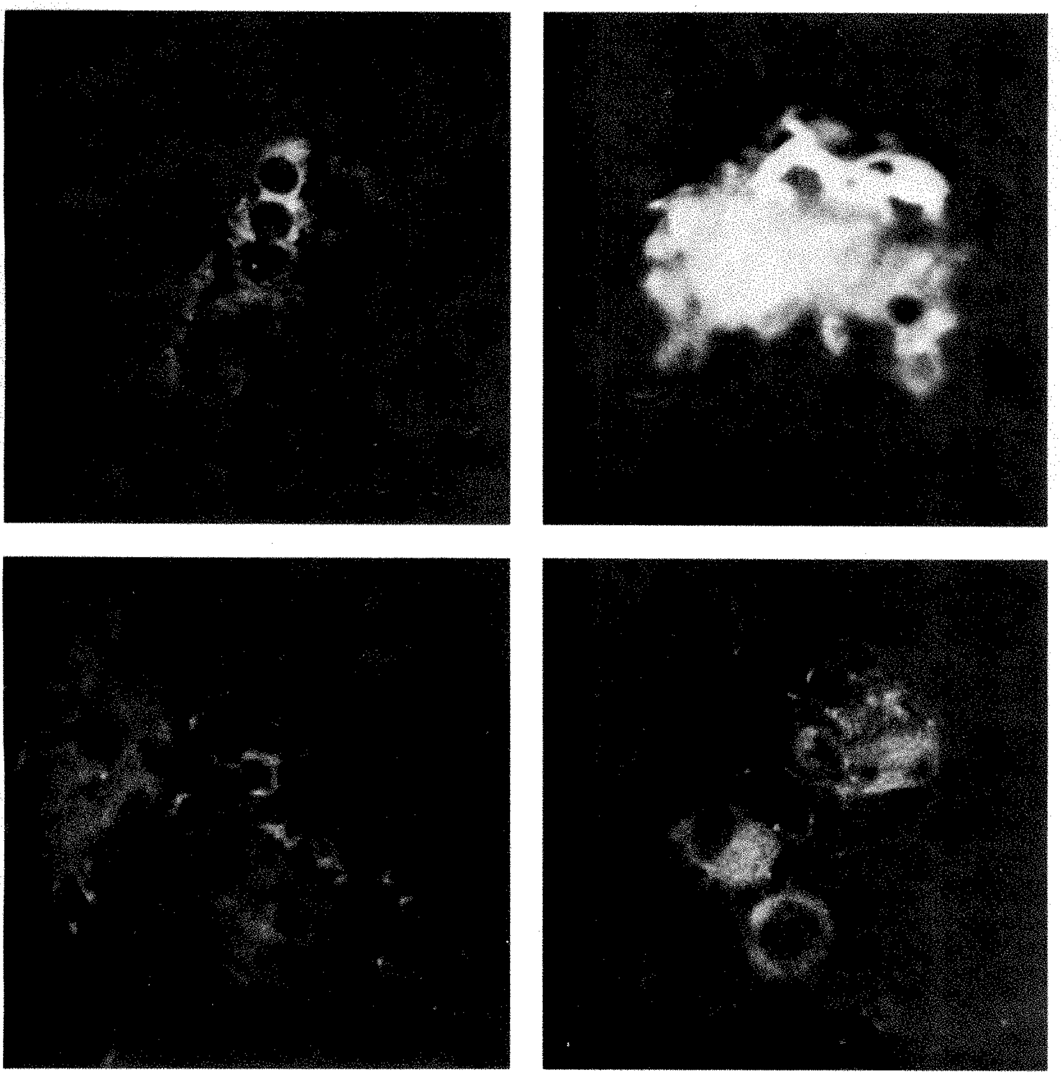

Fig. 1 (Foster and Allansmith). Top left, Corneal endothelial cells from blood type A patient. Fluoresceinated human anti-A antiserum used for staining. Note lack of positive fluorescence (background only, identical to picture obtained after staining with control antiserum). Top right, Corneal epithelial cells from blood type A patient. Fluoresceinated human anti-A antiserum used for staining. Note brilliant fluorescence of epithelial cell membrane. Bottom left, Corneal epithelial cells from blood type A patient. Fluoresceinated control antiserum used for staining. Note lack of fluorescence of epithelial cell membranes. Bottom right, Corneal endothelial cells from the blood type 0 patient. Fluoresceinated human anti-A antiserum used for staining. Note lack of fluorescence of endothelial cell membranes. Identical picture obtained when control antiserum was used for staining (fluorescence microscopy, ×1,000).

showed brilliant fluorescence when the anti-A antiserum was used for staining. No fluorescence was seen when the control antiserum was used on the epithelium from these patients. Both endothelial and epithelial specimens from the type O patients did not show fluorescent staining with either antiserum (Table 2).

Mixed agglutination technique—Blood group specific antigen A could not be found on the corneal endothelial cells of the four type A patients studied with this technique (Table 3) (Fig. 2). Antigen A was easily shown on the corneal epithelial cells of these type A patients with this technique. When anti-A antiserum was applied to these epithelial cells, blood type A (but not type O or type B) indicator cells formed rosettes and multilayered red blood cell aggregates about the epithelial cells. This did not occur if control antiserum was used instead of anti-A antiserum. Both endothelial and epithelial specimens from the four type O patients were negative.

TABLE 2

RESULTS OF FLUORESCENT ANTIBODY STAINING OF HUMAN CORNEAL EPITHELIUM AND ENDOTHELIUM FOR BLOOD GROUP A ANTIGEN*

Blood	No. of	Epithelium		Endothelium	
Group	Subjects	AntiA	Control	AntiA	Control
O	2	0/2	0/2	0/2	0/2
A	3	3/3	0/3	0/3	0/3
AB	1	1/1	0/1	0/1	0/1

*Data expressed as number positive over number tested.

TABLE 3

RESULTS OF MIXED AGGLUTINATION REACTIONS OF HUMAN CORNEAL EPITHELIUM AND ENDOTHELIUM AND BLOOD GROUP A RED BLOOD CELLS*

Blood	No. of	Epithelium		Endothelium	
Group	Subjects	AntiA	Control	AntiA	Control
O	5	0/5	0/5	0/5	0/5
A	4	4/4	0/4	0/4	0/4

*Data expressed as number positive over number tested.

DISCUSSION

Using two separate techniques, different from that used by Herold,[2] we have been unable to detect blood group antigen A on the corneal endothelium of human corneas from blood type A donors. This is important because of the conflicting reports regarding the importance of ABO compatibility to corneal transplant rejection in recipients with vascularized corneas; because of the technical difficulties of detecting the fragile ABO antigens; and because the question of whether one of the major human transplantation antigen systems, the ABO system, is represented on corneal endothelium is crucial to the understanding of which antigenic determinants on corneal endothelium stimulate an immune response. Using the indirect immunofluorescence technique, Herold[2] showed blood group antigens A and B on human corneal epithelium, but could not detect these antigens on Bowman's membrane, stroma, Descemet's membrane, or endothelium. We have now confirmed these findings by using direct immunofluorescence and the ultrasensitive mixed agglutination reaction, two separate techniques, which are different from the technique used by Herold. Szulman[12] pointed out that A antigen deteriorates from a specimen within three to six hours if left in situ (as, for example, awaiting autopsy) and within 12 hours if stored at 4°C (as a surgical specimen, for example). One might argue that Herold failed to detect ABO antigens on corneal endothelium because he used eye bank eyes up to ten hours after death. Our tissue was processed fresh or was frozen at −70°C, and the results were identical to those of Herold.

We believe that the ABO blood group antigens can play no direct role in specific endothelial rejection, because they are not present on human corneal endothelium.

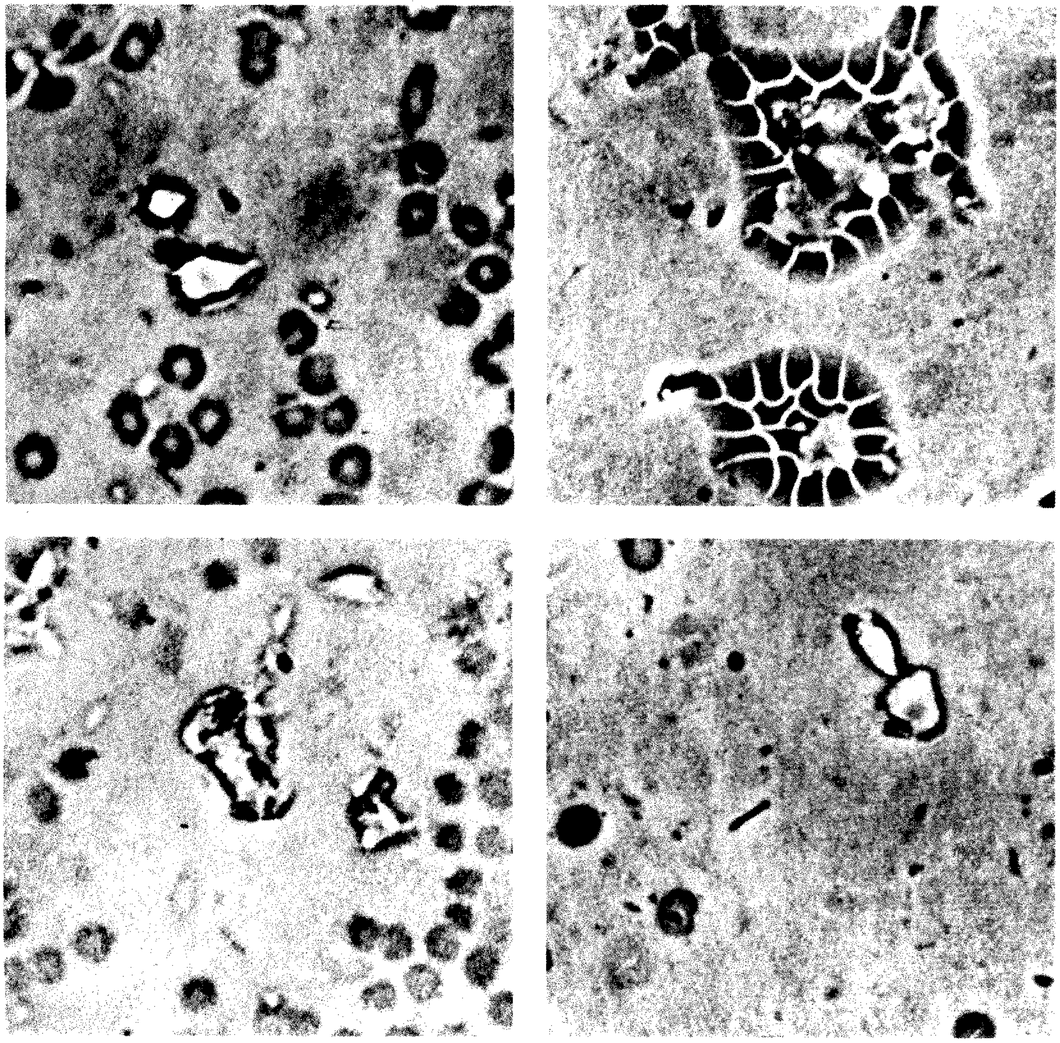

Fig. 2 (Foster and Allansmith). Top left, Corneal endothelial cells from blood type A patient. Slide has been treated with anti-A antiserum followed by washing and application of blood type A human red blood cells. Note lack of adsorption of red blood cells onto endothelial cell membranes. Top right, Corneal epithelial cells from blood type A patient. Slide has been treated with anti-A antiserum followed by washing and application of blood type A human red blood cells. Note dense pack and clustering of red blood cells in rosette formation around the epithelial cells. Bottom left, Corneal epithelial cells from blood type A patient. Slide has been treated with control antiserum followed by washing and application of blood type A human red blood cells. Note lack of adsorption of red blood cells onto epithelial cell membranes. Identical picture obtained if these epithelial cells were treated with anti-A antiserum followed by washing and application of blood type B or blood type 0 red blood cells. Bottom right, Corneal endothelial cells from blood type 0 patient. Slide has been treated with anti-A antiserum, followed by washing and application of blood type A human red blood cells. Note lack of adsorption of red blood cells onto endothelial cell membranes. Identical pictures obtained when blood type O and blood type B indicator red blood cells were used and when control antiserum was used (phase contrast microscopy, ×500).

SUMMARY

Using direct immunofluorescence and mixed agglutination, we did not find blood group antigen A on human corneal endothelium from patients of blood type A or AB. The antigen was easily detected on the epithelium of these patients. We believe that the ABO blood group antigens are probably absent on human corneal endothelium, therefore they cannot play a direct role in specific corneal endothelial rejection.

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NUMBER AND TYPE OF INFLAMMATORY CELLS IN CONJUNCTIVA OF ASYMPTOMATIC CONTACT LENS WEARERS

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We have described a syndrome that affects wearers of both soft and hard contact lenses and consists of papillae greater than 1 mm in diameter, erythema of the conjunctiva, mucus, itching, and decreased lens tolerance.[1] Scanning electron microscopy of the surface of the giant papillae revealed distortion of the cells, centralization of the microvillae, and in general a disturbed surface pattern as compared to normal conjunctivae.[2] The surface characteristics by scanning electron microscopy of the upper tarsal conjunctiva in asymptomatic lens wearers differed from the normal appearance, but resembled some of the changes observed in lens wearers with giant papillary conjunctivitis, in whom areas of normal conjunctiva were also present.[3] Scanning electron microscopy alone did not enable us to determine whether the surface changes seen in the asymptomatic lens wearers were caused by trauma or whether they represented early aspects of the giant papillary process. By using quantitative histologic techniques, we found that all patients with giant papillary conjunctivitis had one or more of the following: (1) mast cells in the epithelium, (2) eosinophils in epithelium or substantia propria, (3) basophils in epithelium or substantia propria. It then became pertinent to determine whether the cellular characteristics of giant papillary conjunctivitis were present in the tissues of asymptomatic contact lens wearers. We report herein the quantitative histologic study of eight asymptomatic contact lens wearers and compare the differential cell counts to cell counts both in normals and in patients with giant papillary conjunctivitis.

SUBJECTS AND METHODS

Subjects—Tissue was taken from the upper tarsal conjunctiva of eight asymptomatic contact lens wearers under regional block anesthesia with 2% block lidocaine (Xylocaine). As reported previously,[3] these individuals all had surface changes distinguishing them from normals and from patients with giant papillary conjunctivitis. The personal characteristics of the eight individuals are shown in Table 1. Biopsy specimens were taken also from 15 persons who never had worn contact lenses and who had white and quiet eyes. These normal control subjects have also been described previously.[4] Quantitative counts of inflammatory cells from 15 individuals with contact

TABLE 1

CHARACTERISTICS OF ASYMPTOMATIC CONTACT LENS WEARERS

Patient No.	Age (yrs)	Lens Type*	Years Worn
1	62	HCL	15
2	27	SCL	5
3	30	HCL	6
4	28	HCL	12
5	23	SCL	1
6	39	SCL	3
7	29	SCL	2½
8	30	SCL	3

*HCL signifies hard contact lens; SCL, soft contact lens.

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lens-induced giant papillary conjunctivitis[5] and qualitative counts from an additional 40 patients with this syndrome were also used. The personal characteristics of normals and those with giant papillary conjunctivitis are included in Table 1. Both hard and soft contact lens wearers were included in the giant papillary conjunctivitis group.

Histologic method—Biopsy specimens were processed in a manner that would maximally preserve basophils, which cannot easily be identified in routine histologic sections, as well as other cell types.[6] Tissues were fixed for three hours in a solution of 2% paraformaldehyde, 2.5% glutaraldehyde, and 0.025% $CaCl_2$ in 0.1M cacodylate buffer, pH 7.4. The fixative was then replaced with cold 0.1M cacodylate buffer, pH7.4, containing 10% sucrose. The tissues were postfixed in osmium tetroxide, dehydrated in ethanol, and embedded in Epon. Sections 1 μm thick were cut, stained with alkaline Giemsa in 2% sodium borate, and examined with a light microscope. This technique is ideal for quantitative analyses of ocular infiltrates because it affords optimal light microscopy structure on large blocks of tissue.

Cell counts—Six cell types were counted: neutrophils, lymphocytes, plasma cells, eosinophils, mast cells, and basophils, according to criteria published previously.[4]

Separate cell counts were made for the epithelium and the substantia propria of each specimen. All areas of each biopsy specimen were counted. Counts were made on two adjacent sections for each biopsy specimen and averaged. If the two counts differed by more than 10%, a third section was counted and the three counts averaged. It was rarely necessary to count a third section.

Calculation of cells per cubic millimeter—Section areas were determined by making a photographic print of each section and tracing the area of epithelium or substantia propria with a planimeter. Cell counts per section were converted to cell counts per cubic millimeter by the following formula:

$$\text{Cells/mm}^3 = \frac{\text{Cells/section}}{\text{Area of sections (mm}^2\text{)}} \times \frac{1}{\text{Average diameter of cell (mm)}}.$$

As the sections were 1 μm thick, the number of sections in 1 mm was 1,000. Calculated cell counts per cubic millimeter were rounded to the nearest thousand.

Statistics—Student's *t*-test was used to compare number of cells in the asymptomatic group with that of normals. P values of $< .01$ were considered significant.

RESULTS

All measurements of number and type of cells for the eight asymptomatic contact lens wearers were within normal limits ($P > .01$) (Table 2).

No eosinophils, mast cells, or basophils were found in the epithelium of the eight asymptomatic contact lens wearers. The distribution of cells within the epithelium and the substantia propria was within normal limits (Figure).

DISCUSSION

The cellular abnormalities in giant papillary conjunctivitis included: (1) mast cells in the epithelium, (2) eosinophils in either epithelium or substantia propria, and (3) basophils in epithelium or substantia propria. The tissues from all 55 individuals with contact lens-associated giant papillary conjunctivitis had one or more of these abnormalities, whereas none were present in the 15 normal subjects. By these criteria, the asymptomatic contact lens wearers have none of the diagnostic abnormalities, and thus no histologic aspect of giant papillary conjunctivitis.

Because by scanning electron microscopy, the conjunctival surface of these eight contact lens wearers differed from

TABLE 2

NUMBER OF INFLAMMATORY CELLS/MM^3 IN UPPER TARSAL CONJUNCTIVA OF ASYMPTOMATIC CONTACT LENS WEARERS

Subject No.	Total Cells	Neutrophils	Lymphocytes	Plasma Cells	Eosinophils	Mast Cells	Basophils
			Epithelium				
1	75,833	12,745	63,088	0	0	0	0
2	3,972	0	3,972	0	0	0	0
3	26,513	694	25,819	0	0	0	0
4	35,067	16,000	19,067	0	0	0	0
5	39,697	3,947	35,750	0	0	0	0
6	17,341	2,128	15,213	0	0	0	0
7	47,086	2,143	44,943	0	0	0	0
8	14,234	1,351	12,883	0	0	0	0
Mean	32,468	4,876	27,592	0	0	0	0
S.D.	22,558	6,038	19,387	0	0	0	0
Upper limit of normal	56,000	26,000	46,000	0	0	0	0
			Substantia Propria				
1	478,635	794	287,135	180,317	0	10,387	0
2	95,812	0	47,617	43,333	0	4,812	0
3	460,666	2,000	386,100	59,733	0	12,833	0
4	101,458	4,516	43,361	51,097	0	2,484	0
5	304,815	1,571	211,131	85,864	0	6,249	0
6	210,798	9,574	196,245	1,702	0	3,277	0
7	181,411	1,327	137,305	41,416	0	1,363	0
8	90,207	593	53,978	33,201	0	2,435	0
Mean	239,350	2,547	165,234	62,083	0	5,480	0
S.D.	159,083	3,148	130,221	53,350	0	4,127	0
Upper limit of normal	410,000	8,000	337,000	100,000	0	11,000	0

that of normals[3] as well as patients with giant papillary conjunctivitis, and the quantitative histology did not establish cellular abnormalities in cases of giant papillary conjunctivitis, the surface abnormalities in these lens wearers were probably caused by trauma. This conclusion could not be drawn, however, from merely looking at and comparing the conjunctival surface in these three groups of eyes by scanning electron microscopy. Months or even years of trauma (caused by the upper eyelid rubbing over either a hard or soft contact lens) would not alone induce the abnormal presence and location of mast cells, basophils, and eosinophils. Because eosinophils and basophils are related to inflammatory processes of immunologic origin, the coincidence of these cell types supports but does not prove the contention that an immunologic process is occurring in the tissue of those with contact lens-induced giant papillary conjunctivitis. The absence of cellular abnormalities in the traumatized asymptomatic conjunctiva renders trauma an unlikely cause of giant papillary conjunctivitis.

SUMMARY

Biopsy specimens were taken from the upper tarsal conjunctiva of eight asymptomatic contact lens wearers and quantitative histology was determined and compared to histology from 15 normals and 55 patients with contact lens-induced

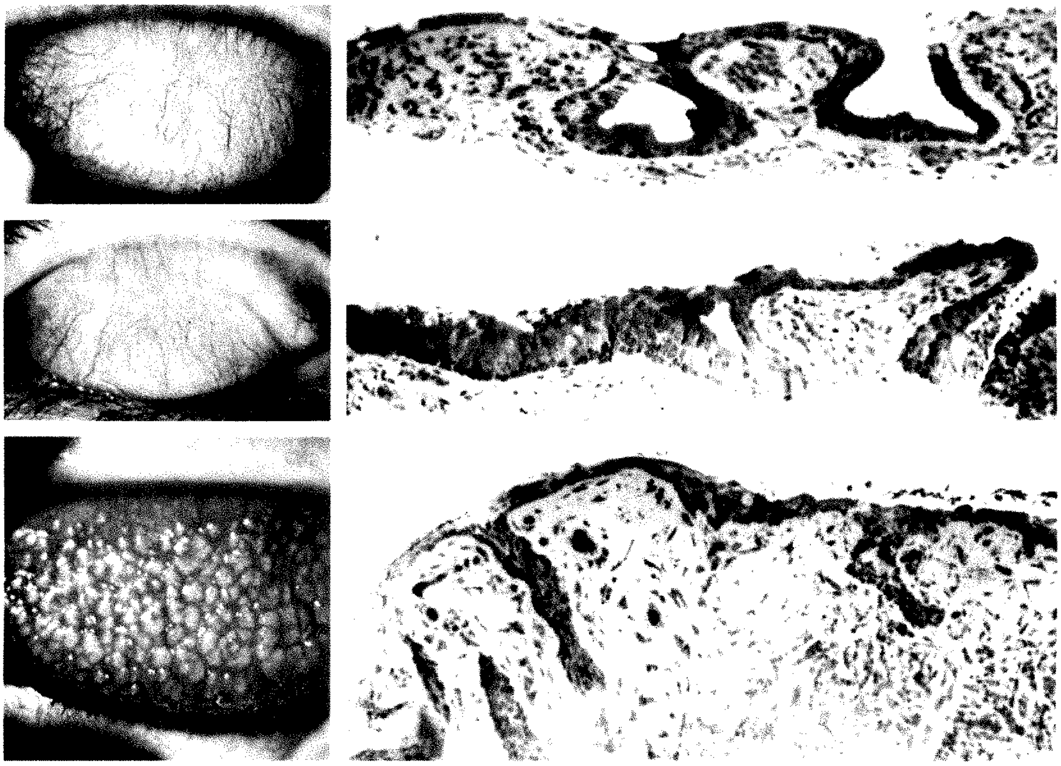

Figure (Allansmith, Baird, and Greiner). Clinical photographs (left) and low-power photomicrographs (×10) (right) of upper tarsal conjunctiva in normal subject (top), asymptomatic contact lens wearer (middle), and contact lens-associated giant papillary conjunctivitis patient (bottom). Note similarity of conjunctivae in top and middle views and giant papillae and thickened conjunctiva in bottom photograph.

giant papillary conjunctivitis. No abnormalities were detected in the asymptomatic contact lens wearers. One or more of the three tissue characteristics of giant papillary conjunctivitis—(1) mast cells in the epithelium, (2) eosinophils in epithelium or substantia propria, and (3) basophils in epithelium or substantia propria—was found in all patients with this syndrome, but none was present in any of the eight asymptomatic lens wearers.

Trauma occurring over months or even years does not induce histologic alteration in the upper tarsal conjunctiva; the surface changes seen previously by scanning electron microscopy in asymptomatic lens wearers are secondary to trauma, not early aspects of the giant papillary conjunctivitis syndrome.

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INTERSTITIAL KERATITIS CAUSED BY AMERICAN (MUCOCUTANEOUS) LEISHMANIASIS

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American leishmaniasis is a disease caused by the flagellate protozoa, *Leishmania braziliensis,* which was observed in 1909 by Lindenberg[1] and Carini and Paranhos[2] in smears of patients with Bauru ulcer in São Paulo, Brazil. In 1911, Vianna[3] studied this type of leishmania, considered it a new species and named it *L. braziliensis.* The disease occurs in the areas from southern Mexico to northern Argentina and is extensive in Brazil and Peru.[4]

The *Leishmania* (*L. brasiliensis*, producing American leishmaniasis; *L. donovani*, producing kala-azar; and *L. tropica,* producing Oriental sore) has two stages of development: the aflagellate stage occurring in man and other vertebrates and the flagellate stage occurring in insect vectors and culture media.

The vector, the mosquitoes of the genus *Phlebotomus*, are small hairy mosquitoes that acquire the infection by ingesting the parasite when taking a blood meal from an infected host. The parasite is transmitted to a healthy human or other vertebrate host by the bite of the infected female mosquito.

The extra-human reservoirs are wild rodents, including rats, mice, agoutis, and pacas, which are common in the neotropics.

The incubation period varies from two weeks to two months, after which an erythematous papule develops at the site of inoculation and gradually increases in size by peripheral extension. The papule may ulcerate, vesiculate, or take on a mulberry aspect. Regional adenopathy may be noted. Generally, the lesions are secondarily infected.

The cutaneous lesions have an infiltrated raised border and the interior of the sore is often covered by a serosanguinous exudate or yellow membrane. A lymphatic form may be seen simulating sporotrichosis extending along the lymph channels, manifested by thickening of the lymph vessels, nodules lymphangitis, and adenitis.

Mucous membrane lesions differentiate American leishmaniasis from Oriental sore. The mucous membrane lesions almost always begin in the nasal mucosa, which becomes inflamed, edematous, and finally ulcerative. The nasal cartilage usually is invaded and destroyed, resulting in a deformity known as "tapir's nose," "camel's nose," or "parrot's beak." The upper lip becomes thickened and protrudes forward, accentuating the deformity. The involvement of nasal fossae, pharynx, soft palate, floor of the mouth, tonsils, and tonsilar fauces, as well as the respiratory tract, pharynx, and larynx may cause difficulty in breathing, feeding, and deglutition. As a result of acute respiratory infection, malnutrition and death may occur.

Disseminated anergic leishmaniasis as manifested by specific anergy to leismanin antigen (Montenegro's antigen), absence of visceral involvement but widespread involvement of the skin, probably from hematogenous dissemination, may occur.

Ocular complications caused by American leishmaniasis have been reported,[5-25]

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but reports of interstitial keratitis as a result of American leishmaniasis are uncommon,[5,6,9,13,18,26] and I am unaware of any report in the past 25 years. I report herein a case seen at the Eye Clinic of the Hospital das Clínicas Medical Faculty, São Paulo University.

CASE REPORT

A 58-year-old man came to the eye clinic complaining of decreased vision in both eyes, and sores in the nose of 20 years' duration (Fig. 1). The patient had been bitten by a mosquito behind the right ear 35 years previously. Treatment with antimonial drugs apparently resulted in cicatrization of the ulcer, but ten years later, new sores appeared that involved his nose, and over the past two years the sores became much worse. The patient noted photophobia, tearing, hyperemia, and decreased vision in both eyes for over 20 years.

Examination revealed visual acuity was reduced to finger counting at 2 m in the right eye and 20 cm in the left eye, which did not improve with lenses or pinhole. The limbal conjunctiva was hyperemic. A diffuse leukoma and cellular infiltration involved the entire cornea of both eyes, together with diffuse vascularization of the superficial stroma. The epithelium was intact (Fig. 2). The lens, iris, and fundus were poorly visible but appeared to be grossly normal.

The general examination revealed diffuse enlargement of the nose with ulcers in various stages of development and purulent discharge. The nasal septum was absent. Ulcers occurred in the patient's gums, tonsillar pillae, and the left malar region. There was a cicatrix behind the right ear in the spot where the patient said the mosquito had bitten him.

Laboratory studies included Montenegro test (positive at 48 hours); indirect immunofluorescence for anti-*Leishmania* antibodies (positive at 1:10); VDRL and FTA-ABS (negative); Mantoux test (negative at 1:1,000 dilution); and chest x-ray film (normal). Cultures of the conjunctiva and cornea for fungi, bacteria and parasites, including cultures on Nicole, Novy, McNeal medium, were negative.

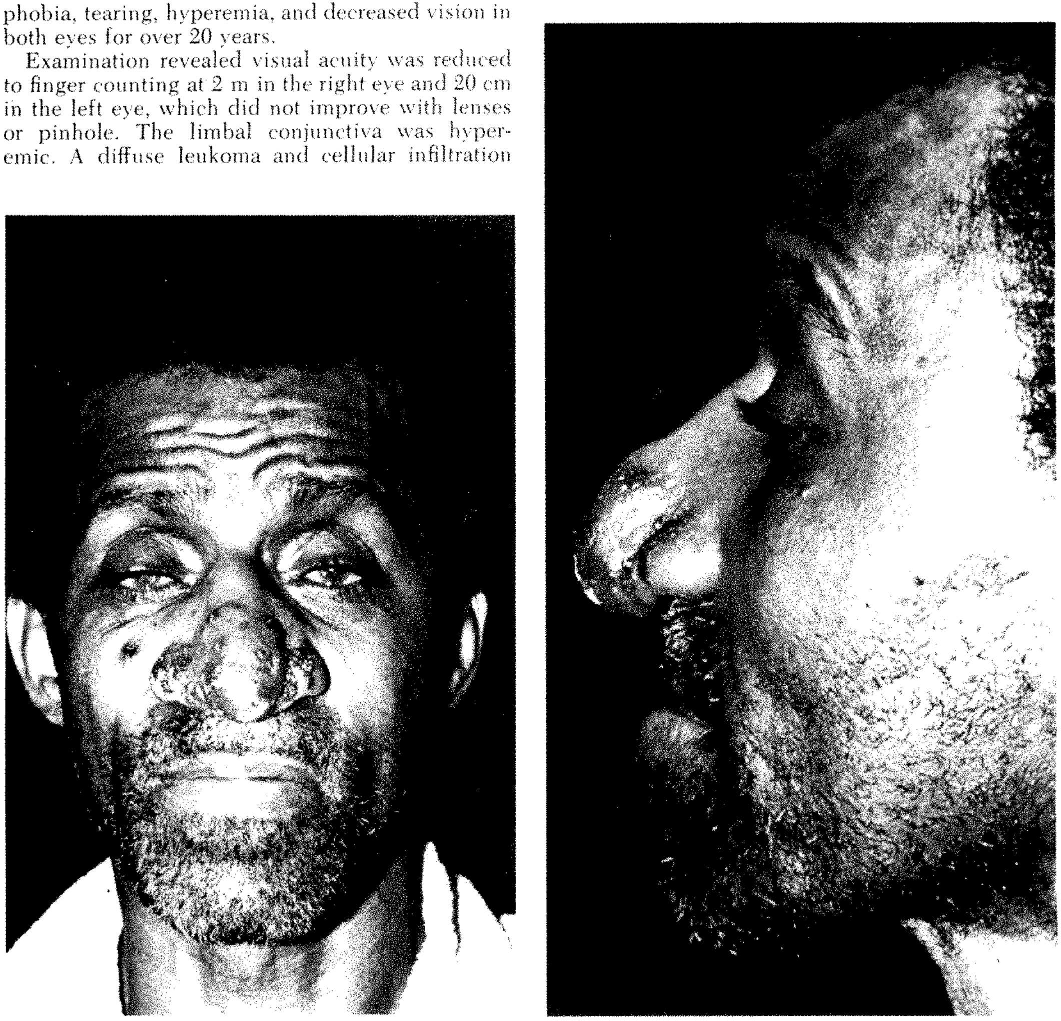

Fig. 1 (Roizenblatt). Front view (left) and side view (right) of facial aspects of patient before treatment.

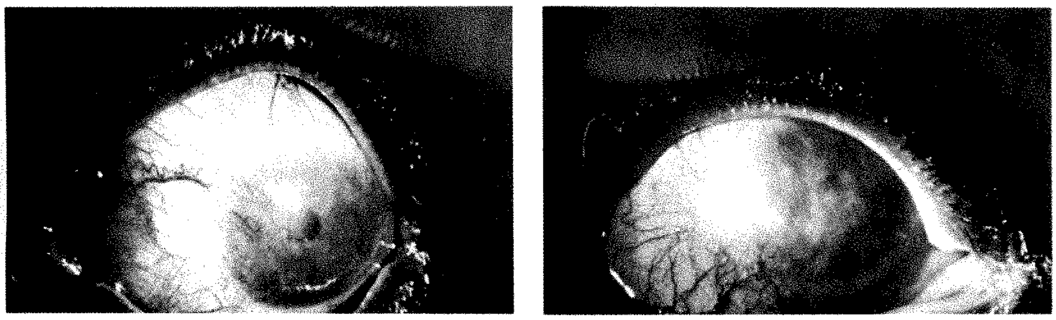

Fig. 2 (Roizenblatt). Corneal (left) and conjunctival (right) aspects of the right and left eyes.

Scrapings of the bulbar conjunctiva and cornea including studies with the Leishman dye and acid-fast stain were negative.

A biopsy specimen of the nasal lesions showed hyperkeratosis, corneal pearls of the epidermis, and atrophy and hydropic degeneration of the basal cell layer and pigmentary drop off into the dermis. Perivascular round cell infiltration was noted in the dermis, together with vacuolation of the histiocytes (Fig. 3).

Biopsy of the conjunctiva showed exfoliation of the epithelial layer, round cell infiltration in the stroma, and tuberculoid granulomas without caseation (Fig. 4).

Treatment with amphotericin B (total dose of 1 g) was given and the ulcerative areas showed marked improvement (Fig. 5).

DISCUSSION

Although we were unable to identify the parasite in this patient, the clinical, laboratory, and pathologic findings and the therapeutic response suggest that the patient was suffering from American leishmaniasis. Many authors believe that the lack of finding a parasite in the lesion does not rule out the diagnosis.[7,16–18,27]

Epidemiologically, the patient was born in an endemic area for leishmaniasis (Bahia), and at the time of the original ulcer formation, he was working as a farmer in another area endemic for leishmaniasis (Marilia, São Paulo).

The Montenegro test, positive in this patient, is positive in more than 90% of patients with the disease.[4,7,18,20] It has certain limitations in endemic zones, as patients may have a primary infection with *Leishmania*, which evolves spontaneously and thus gives a positive response.

Differential diagnosis—The dermatologic aspects of this patient suggest various diagnoses, but we are unable to

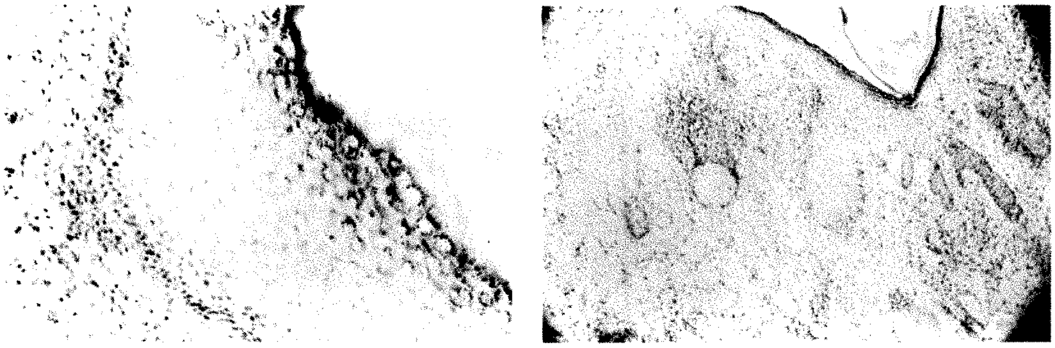

Fig. 3 (Roizenblatt). Biopsy specimen of the nasal lesion showing hyperkeratosis and corneal pearls of the epidermis together with atrophy and hydropic degeneration of the basal cell layer and pigmentary drop off into the dermis. Perivascular round cell infiltration is present in the dermis, together with vacuolation of the histiocytes (×100 and ×400).

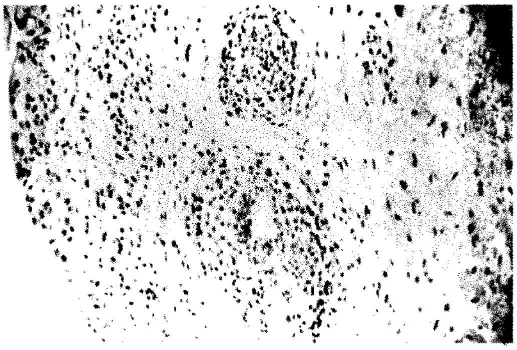

Fig. 4 (Roizenblatt). Biopsy specimen of the conjunctiva showing exfoliation of the epithelial layer, round cell infiltration in the stroma, and tuberculoid granulomas without caseation.

establish evidence of fungal infections such as paracoccidioidomycosis, sporotrichosis, histoplasmosis, and chromoblastomycosis, or bacterial infections such as rhinoscleroma, tuberculosis, or leprosy on culture or smear.

The ocular aspects of the patient suggest various other diseases, including trachoma, syphilis, tuberculosis, leprosy, trypanosomiasis, and onchocerciasis.

Trachoma was not considered a likely cause, as the vascularization occurred in the superficial stroma and did not resemble the pannus seen with trachoma. Additionally, there was no supporting evidence of trachoma, such as follicles, conjunctival scarring, trichiasis, or Herbert's pits.

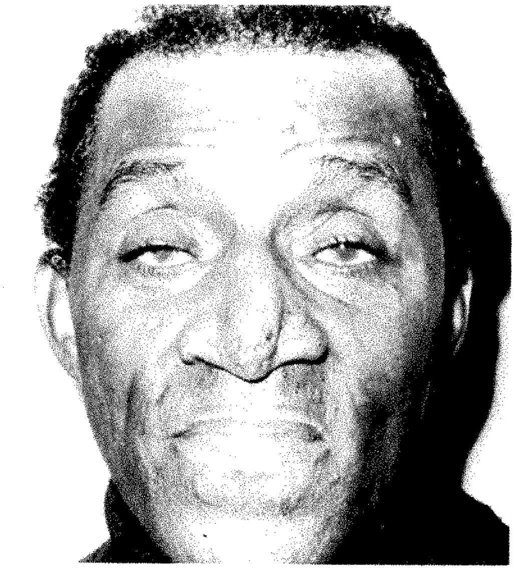

Fig. 5 (Roizenblatt). Facial aspect of patient after treatment.

The vascularization of the cornea and the inflammatory reaction associated with syphilis is predominate in deep stroma, in contrast to the superficial inflammation in this case. Additionally, there were no other signs of congenital syphilis, such as Hutchinson teeth, raspberry molars, saddle nose, or deafness, and the serologic tests virtually ruled out the probability of syphilis.

Tuberculosis must be considered in the differential diagnosis of interstitial keratitis; however, the patient was anergic to tuberculin, the chest x-ray film was negative, and the biopsy specimen showed no caseation.

Lepromatous leprosy may cause a superficial vascularization of the cornea together with skin changes, somewhat similar to that noted in this patient, but smears and biopsy usually show the specimen teeming with organisms.

Trypanosimiasis and onchocerciasis may cause corneal vascularization, but these diseases are not seen in the areas in which the patient lived, and the organisms were not seen on biopsy.

Other causes of interstitial keratitis including herpes simplex keratitis (herpes simplex is the great imitator), lymphogranuloma venereum (which may cause a segmental sclerokeratitis), and mumps (which do not cause vascularization of the corneal stroma) have not been ruled out as the cause of the ocular lesions, but their presence is unlikely, and they would not cause the skin lesions.

American leishmaniasis has various forms, including cutaneous American leishmaniasis, mucocutaneous American leishmaniasis, disseminated anergic leishmaniasis, and Mexican leishmaniasis. The present case falls into the category of mucocutaneous American leishmaniasis.

Ocular findings associated with mucocutaneous American leishmaniasis include involvement of the eyelids[6,8,9,11,17–19,26,28] and conjunctiva,[10,13,15,18,23,25–27] described as eyelid edema; scarring or even destruction of the tarsus; and nodular granulomas of the tarsal or bulbar conjunctiva. Ulceration of the conjunctiva and cornea and ulcerative[5,6,18,25–27] or granulomatous[5,10,16,18,24–26] lesions of the cornea, as well as interstitial keratitis[5,6,9,13,18,26] also have been described.

The eye may be involved by contiguous spread to the eyelid and conjunctiva, by hematogenous spread, or by inoculation of the conjunctiva by the patient's own fingers.[5,6,13,15,17,18,20,26] In our patient, the most likely spread was by way of the blood stream, as there were no signs of tarsal or conjunctival involvement.

The disfigurement and lack of satisfactory treatment have often hampered case findings of patients with leishmaniasis, and when found, little attention has been paid to the ocular findings. Case findings and ophthalmologic examination would reveal many more cases than the reports suggest.

SUMMARY

A 58-year-old man with American (mucocutaneous) leishmaniasis had the unusual ocular complication of interstitial keratitis. Although the parasite was not identified, clinical laboratory and histologic findings were compatible with leishmaniasis, and tests ruled out other causes. Ulcerative areas improved after treatment with 1 g of amphotericin B.

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INFECTION AFTER SPONGE IMPLANTATION FOR SCLERAL BUCKLING

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Responding to criticism over potential infection with the use of silicone sponge for scleral buckling, we reported on the incidence of infection at New York Hospital.[1] There had been 27 infections in 800 operations, an incidence of 3.5%. Since then, this figure has been contradicted and confirmed. Russo and Ruiz[2] reported that it was necessary to remove 31 of 127 sponge explants of which 23 were infected, an incidence of 18%. Ulrich and Burton[3] had an infection rate of approximately 7% (9) in 132 sponge operations. McPherson and Moura[4] reported infections requiring removal of the sponge in 3.8% (19) of 503 patients with sponges.

We report herein on infections in 1,900 sponge operations done at the New York Hospital and the University Eye Clinic in Bonn, West Germany, between 1970 and 1976. The infection rate from all organisms including coagulase-negative *Staphylococcus* was 3.4% (31/900) in New York and 2.7% (27/1,000) in Bonn. Coagulase-negative *Staphylococcus* continues to be the most frequently recovered organism and there is confirming evidence that it acts as a pathogen in the presence of silicone implants.[5-7] Infection with coagulase-positive *Staphylococcus* has diminished to 0.2%, which compares with the figure (0.6%) in McMeel's report[8] on infections caused by coagulase-positive *Staphylococcus* using solid silicone implants.[8]

MATERIAL AND METHODS

We analyzed from New York the summation of data collected between 1970 and 1976 from 900 consecutive operations, and from Bonn we analyzed data from 1,000 consecutive operations done during the same period.

The methods at the two hospitals were almost identical. Both used preoperative local antibiotics and tried to provide an aseptic operative field described previously.[1] In the operations done at Bonn, the extra precaution of doing a smear of conjunctiva on the morning of surgery was taken. If bacteria were present, the operation was delayed until the conjunctiva was bacteria free.

The operative technique for both was a modified Custodis operation using cryopexy to the edges of the break and a Silastic sponge explant compressed over full-thickness sclera.[9,10] In New York a culture was taken of the buckle site in the middle of the operation to provide a profile of bacterial contamination.

Both services soaked their sponges in parenteral concentration of antibiotic solutions and squeezed the sponges to absorb some of the solution. The remainder of the antibiotic solution was then injected into Tenon's space after closure. The most significant difference was in the postoperative use of antibiotics. In New York, aside from local application of bacitracin ointment to the eyelid margin at the conclusion of surgery, no antibiotics were used. In Bonn the patients were treated with 4,000,000 units of penicillin daily

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TABLE 1

PREDOMINANT SIGNS FOR 31 BUCKLES REMOVED IN NEW YORK

Type	No. of Patients	Average Time of Onset	Range
Fistula	9	1.4 mos	2 to 16 wks
Granuloma	14	11 mos	1 mo to 5 yrs
Hemorrhagic tumor	7	15 mos	1 mo to 4 yrs
Exposure	1	1.5 yrs	1.5 yrs
Total	31	9 mos	2 wks to 5 yrs

for five days, and given local antibiotics for three weeks.

RESULTS

Of 900 scleral buckles in the New York series, 31 had to be removed for infection or exposure. In Bonn 27 of 1,000 were removed.

The results of buckle infection are presented in Tables 1 and 2. Primary infection of the explant manifested as a fistula, granuloma, or a subconjunctival hemorrhagic mass. Fistulas occurred relatively early in the postoperative period and were caused by virulent organisms such as coagulase-positive *Staphylococcus*, and *Proteus mirabilis*. Granulomas occurred later and were caused by *S. epidermis*. Subconjunctival tumors also appeared and proved to be infected granulomas made evident by hemorrhage.

In the Bonn series exposure of the explant occurred in the first and second weeks and was attributed to inadequate closure. The one exposure in a New York patient occurred after 18 months and was caused by inadequate scleral suturing; the suture bite was too short or too superficial and pulled out. As a result, the explant migrated anteriorly, and a Dacron knot on the external surface eroded through the conjunctiva (Fig. 1). Infection was secondary to exposure.

The intraoperative cultures obtained from 400 buckle operations done in New York between 1973 and 1976 were collated for information on bacterial contamination in the operative field (Table 3). The field was contaminated 59% of the time; 48.5% of the time with coagulase-negative *Staphylococcus,* 2.75% with coagulase-positive *Staphylococcus*, and 3% of the time with *Proteus.* The bacterial percentages were strikingly similar to those obtained from 400 operative cultures obtained between 1966 and 1969,[1] and indicated that the bacterial flora at New York Hospital had not changed in a decade.

Staphylococcus epidermis was the organism that most frequently caused clinical infection with a silicone explant operation. It was found in 21 of 31 buckles removed in New York and in eight of 19 buckles from which cultures were available in Bonn (Table 4). In Bonn there was

TABLE 2

PREDOMINANT SIGNS FOR 27 BUCKLES REMOVED IN BONN

Type	No. of Patients	Average Time of Onset	Range
Fistula	5	1 mo	0.5 to 2 mos
Granuloma	10	18 mos	1 mo to 7 yrs
Hemorrhagic tumor	7	5 mos	1 to 13 mos
Exposure	5	1.5 wks	1 to 2 wks
Total	27	13 mos	1 mo to 7 yrs

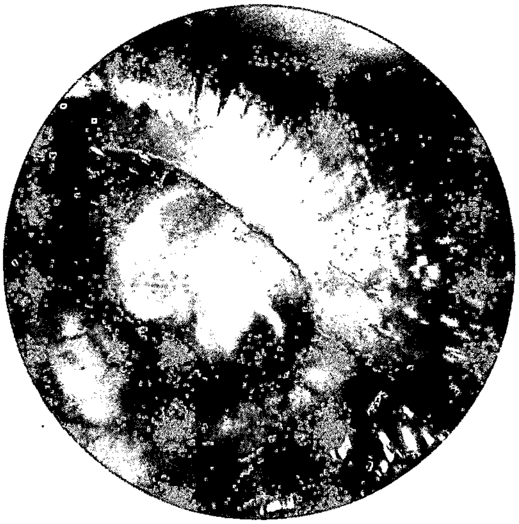

Fig. 1 (Hahn and associates). Migration and exposure of a silicone sponge caused by avulsion of the Dacron suture after 18 months.

no growth from the culture of the sponges nine times and in New York two times, even though the buckle sites were infected. Possibly this was an effect of antibiotic therapy; all the patients in Bonn and one of the New York patients were treated with systemic and local antibiotics before the surgical exploration. It raises the question of whether an infected granuloma can be sterilized. Antibiotics may reduce bacterial count and residual bacteria might have been found if more cultures had been taken.

TABLE 4

CULTURES FROM BUCKLES REMOVED

Organism	No. of Patients New York	 Bonn
S. epidermis, coagulase-negative	21	8
S. aureus, coagulase-positive	2	0
Proteus	3	1
Klebsiella	0	1
Enterobacter aerogenous	1	0
Streptococcus viridans	1	0
No growth	2	9
No report	1	8
Total	31	27

The routine operative culture in New York coincided with the eventual organism of clinical infection 75% of the time, and was evidence that the source of clinical infection was contamination at the buckle operation. The conjunctival culture obtained before removal of the explant coincided with the organism 83% of the time, and was useful in deciding the urgency for removal.

The incidence of infection will vary with the quantity of silicone material included in the operation.[1,3] In the present series, operations that included a single sponge, became infected 1.4% of the time in New York and 1% in Bonn. With multiple sponges and long circumferentials, the rate of infection rises, reaching its maximum with combinations of radial

TABLE 3

RESULTS OF 400 CONSECUTIVE OPERATIVE CULTURES IN NEW YORK (1973 TO 1976)

Organism	1st 100	2nd 100	3rd 100	4th 100	Average (%)
No growth	38	42	52	32	41
S. epidermis	54	50	34	56	48.5
S. aureus	2	2	5	2	2.75
Proteus species	2	3	4	3	3
Corynebacterium species	7	4	6	7	6
Escherichia coli	1	1	1	0	0.75
Streptococcus species	1	1	1	0	0.75
Enterobacter aerogenous	1	0	0	0	0.25
Neisseria species	1	0	0	0	0.25
Candida species	0	0	0	1	0.25

TABLE 5

QUANTITY OF BUCKLE

	Total Patients(%)		Removed(%)	
Buckle	New York	Bonn	New York	Bonn
Single sponge>50°	47	52	1.4	1
Multiple sponges>50°	15	20	3.7	5
Circumferential sponge<50°	25	26	5.3	3.8
Encircling element	13	2	6.8	10
Average	—	—	3.4	2.7

sponges and encircling elements; bands were used in New York and 3-mm encircling sponges were used in Bonn (Table 5).

Patients who have only one operation (84% (758/900) in New York and 89% (890/1,000) in Bonn), regardless of the extent of the operation, became infected 2.3% and 1.9% of the time. Reoperations became infected 9.2% and 9.9% of the time, five times as often (Table 6).

In seven patients some degree of scleral necrosis was observed beneath the explant (Table 7). Necrosis was arbitrarily graded as 1 to 4+; 4+ being exposure of the choroid. The patients were divided into two pathogenetic groups; one in which necrosis was caused by proteolytic enzyme and another in which necrosis was mechanical in origin. In the first group were two patients with short sponges who developed fistulas within a month. Both were contaminated with *Proteus*. The damage beneath the explant was characterized by a discoloration and softening of the sclera, the effect of proteolytic enzyme. The 1+ necrosis attributed to both cases is an estimate of scleral thinning and does not reflect the greater degree of intrinsic damage. In the second group the explants were removed late, between ten months and five years. The scleral damage occurred beneath relatively long circumferential sponges or constricting bands. All were contaminated with *S. epidermis*, and the presence of granuloma was the reason for removing the explant. The type of necrosis found beneath the sponges or bands was clean and sharp and resembled the effect of pressure on the sclera rather than the necrosis from bacterial enzymes.

In New York, four patients had redetachments after the buckles were removed (Table 8). The buckles had been in place more than one but less than four months, and redetachment occurred quickly, between three days and two weeks after the buckles were removed. Two were reattached with another buckling procedure. Two patients developed preretinal proliferation that resisted repair. In one of these the infection, *P. mirabilis*, apparently contributed because the vitreous was infiltrated by a cloud of white cells over the buckle. In the other, also *P. mirabilis*, the infection probably did not contribute because the vitreous remained clear; preretinal proliferation had been present and progressive before the detachment operation. In the Bonn series, none of the patients had detachments after removal of their buckles.

TABLE 6

INCIDENCE OF REMOVAL RELATING TO REOPERATION

	Total Patients(%)		Removed(%)	
No. of Operations	New York	Bonn	New York	Bonn
One	84.3	89	2.3	1.9
Two or more	15.7	11	9.2	9.9

TABLE 7

SCLERAL NECROSIS*

Patient No.	Degree	Duration of Buckle	Organism	Procedure Orientation	Extent	Material
1	1+	1 mo	Proteus	Circumferential	90 degrees	4-mm sponge
2	1+	1 mo	Proteus	Radial	50 degrees	5-mm sponge
3	1+	15 mos	*S. epidermis*	Circumferential	360 degrees	No. 41 band
4	2+	20 mos	*S. epidermis*	Circumferential	210 degrees	5-mm sponge
5	3+	5 yrs	*S. epidermis*	Circumferential	180 degrees	3-mm sponge
6	4+	3.5 yrs	*S. epidermis*	Circumferential	360 degrees	No. 40 band
7	4+	10 mos	*Enterobacter*	Circumferential	360 degrees	No. 40 band

*Seven of 31 patients.

Between 1969 and 1970 there were four infections caused by *S. epidermis*, one caused by *S. aureus*, and one by a less frequent organism, *S. viridans* (Fig. 2). Chloramphenicol was injected into Tenon's space. From 1970 to 1974 lincomycin was used, then clindamycin for almost two years, and gentamycin beginning in 1976. The differences in rate or type of infection was not significant. The absence of infection for two years suggests that this antibiotic may be more effective but not yet statistically significant. McPherson and Moura[4] reported effective results with gentamycin.

DISCUSSION

Clinical infection of silicone sponge buckles with pathogenic organisms such as coagulase-positive *Staphylococcus* and *Proteus* manifests itself early as fistulas of the wound. The toxins of these organisms cause scleral necrosis, can infiltrate the vitreous, and may provoke preretinal retraction. Our claim[1] that scleral abscess and perforation would not occur if cryopexy was substituted for diathermy, must be modified. Although the sclera is more resistant to penetration, and scleral abscess with rapid melting of the scleral wall has not occurred, the softening and discoloration of the scleral wall beneath the explant in the two patients infected with *Proteus* suggests the same process.

Our belief that the coagulase-negative *Staphylococci* act as pathogens in the presence of a silicone explant, is reinforced by similar experience with implantation of silicone structures in heart valve replacement,[5] joint replacement,[6] and mammary prosthesis.[7] The symptoms with scleral explants occur late, on the

TABLE 8

REDETACHMENT AFTER REMOVAL*

Patient No.	Duration of Buckle	Time after Removal	Organism	Vitreous Cloud	Final Result†
1	2,5 mos	2 wks	*S. epidermis*	0	Attached
2	4 mos	2 wks	*S. epidermis*	0	Attached
3	1 mo	3 days	*Proteus*	2+	mpr
4	1,5 mos	2 wks	*Proteus*	0	mpr

*Four of 31 patients.
†mpr designates massive preretinal retraction.

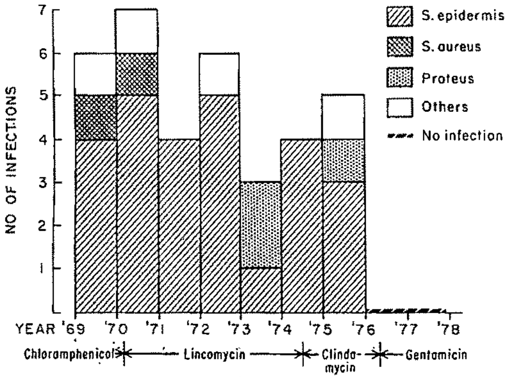

Fig. 2 (Hahn and associates). Infections at New York Hospital over nine years, plotted against antibiotics injected into Tenon's space.

average ten months after operation, and are the manifestations of an enlarging and discharging granuloma. Although visual function is not threatened and antibiotics may be temporarily effective, permanent relief can only be obtained by removing the explant.

SUMMARY

In one series of 900 explant scleral buckling procedures, 31 had to be removed over seven years because of contamination. Of 1,000 explants from a parallel series, 27 had to be removed. In the second series, requirement of a sterile conjunctiva preoperatively and the use of prophylactic antibiotics postoperatively did not significantly reduce the rate of infection.

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FOVEAL HYPOPLASIA AND BILATERAL 360-DEGREE PERIPHERAL RETINAL ROSETTES

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Macular aplasia has been associated with eye disorders such as albinism, aniridia, unilateral myopia, colobomata, and microphthalmos.[1] Cases of isolated foveal hypoplasia without other associated ocular disorders have been reported by Curran and Robb.[2] Congenital peripheral retinal rosettes have been studied in autopsy eyes by Teng and Katzin.[3]

We report the case of a patient with foveal aplasia and a unique distribution of bilateral, 360-degree retinal rosettes. The occurrence of these entities together may be due to an intercurrent infection during the late period of embryogenesis.

CASE REPORT

A 24-year-old woman has had pendular nystagmus since birth and a visual acuity of 6/60 (20/200) in each eye since her initial ocular examination. Refraction was +1.25 − 0.75 × 180 in the right eye and +2.00−1.50 × 180 in the left. Correction did not improve her visual acuity and she had no clear foveal reflex. Normal macular pigmentation was present in the posterior pole of both eyes. In the ten years she has been observed, there has been no significant change in her ophthalmologic status. There was no evidence of mental retardation or deficiencies in her sensorimotor development. She is presently married and has one daughter.

No history of prematurity, or of irradiation during or after gestation was reported. By history, late in the third trimester, the patient's mother and older sister had whooping cough which persisted into the postpartum period. There was no consanguinity between her parents. The patient's family members were given complete eye examinations. The patient's brother had asymptomatic, increased intraocular pressures of 30 mm Hg in the right eye and 32 mm Hg in the left, normal visual fields and gonioscopy examination, and a small peripheral retinal hole. With the exception of the patient's sister, who had a similar retinal hole, no significant ocular abnormalities were found in the rest of her family.

An eye examination revealed that pendular nystagmus was of equal amplitude in all positions of gaze including the primary position. Schiøtz tonometry measurements were 20.1 mm Hg in both eyes. The patient's eyelids, orbit, conjunctiva, cornea, anterior chamber, and pupil were all within normal limits when examined with the slit lamp. She had brown irides without signs of iris aplasia or hypoplasia, there were no cataracts or lens dislocations, and the vitreous appeared normal, without cells, liquefaction, or membrane formation. There was no evidence of abnormal vitreous adhesions or detachments.

Indirect ophthalmoscopy revealed that the optic nerve heads were of normal diameter and slightly tilted nasally. They were both pink, without evidence of optic atrophy or abnormal glaucomatous cupping. The retina appeared grossly normal bilaterally with no loss of pigment epithelium. The vessels of both retinas appeared normal with an artery-to-vein diameter ratio of 2:3. The peripheral fundus was circumscribed bilaterally with yellowish white nodules, approximately 0.2 to 0.5 mm in diameter. These small nodules were spaced 0.5 to 0.7 mm apart in a discrete line on the retina, approximately 2 to 3 mm from the ora serrata. In most regions, they were aligned in a single row (Figs. 1 and 2). Nasally, there was a line two to three nodules wide on the retina.

The ora serrata was poorly developed, 360 degrees bilaterally. No distinctive oral bays or teeth were distinguished. The nasal and temporal ora serrata appeared equally indistinct (Fig. 1).

In the macular region, the foveal reflex was absent. The region was identified only by the presence of the superior and temporal blood vessels converging on the presumed macular region and by normal macular pigments (Fig. 3). The fluorescein angiogram of the fundus revealed a macular hypofluorescence inferior to the normal macular position. The retinal vasculature surrounding both macular regions was unusual; a cilioretinal vessel appeared to cross over the usually vessel-free zone of the macula and over the horizontal raphe (Fig. 4). The patient failed to see the Haidinger brush phenomenon with either eye.

Despite the patient's pendular nystagmus, she was able to moderate the nystagmus in the primary

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Reprint requests to Editorial Services Unit, Eye Research Institute of Retina Foundation, Boston, MA 02114.

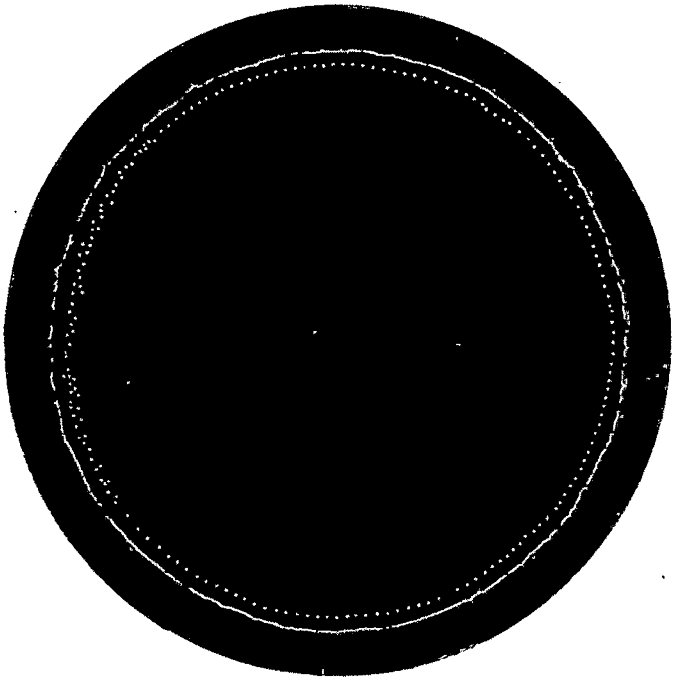

Fig. 1 (Yoshizumi, Thomas, and Hirose). Drawing of the fundus (left eye) of the patient showing retinal rosettes circumscribing 360 degrees of the peripheral fundus. The rosettes formed a single row around most of the peripheral fundus, but formed clusters of two to three nodules nasally from the 8 o'clock to 10 o'clock meridians. There was an absence of the macula's reflex. Note poorly formed oral bays reminiscent of the embryonic fundus.

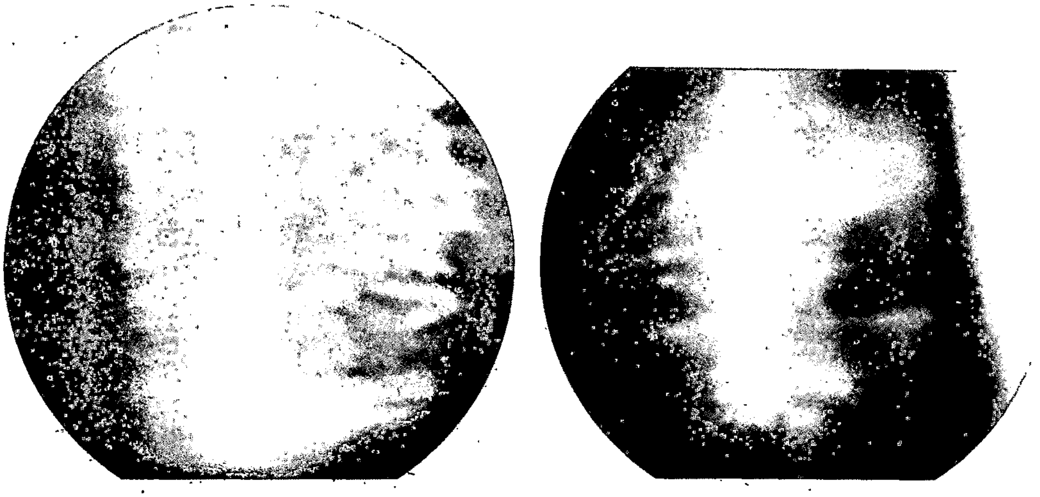

Fig. 2 (Yoshizumi, Thomas, and Hirose). Fundus photograph of retinal rosettes in the periphery. Left, Temporal view. Right, Nasal view; note wider band of rosettes formed by multiple rosettes compared to the single arrangement of rosettes temporally.

position to allow reproducible electroretinogram (ERG), visual-evoked response, and electro-oculogram (EOG) measurements.

An ERG revealed that the photopic and scotopic b-waves and a-waves were of normal amplitudes and peak-time responses were within normal limits. The photopic flicker response was slightly smaller than normal, and the oscillatory potentials were reduced. A visual-evoked-response recording was performed using a 4.5-degree white spot stimulating the macular region of each eye. The amplitudes were small with peak delays in both eyes. These results were consistent with impaired macular function. An EOG revealed that light peak vs dark trough ratios were within normal limits for both eyes.

Visual fields performed by kinetic perimetry, plotted with a 1-mm² and a 64-mm² object under standard background illuminance of 5.32 mL and object

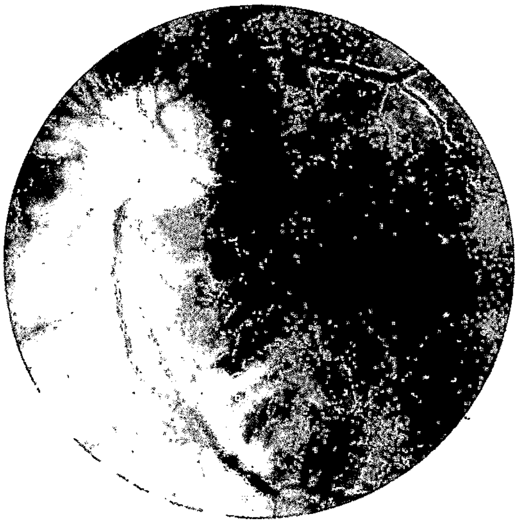

Fig. 3 (Yoshizumi, Thomas, and Hirose). Fundus photograph shows increased pigmentation in the macular region without a clear foveal reflex.

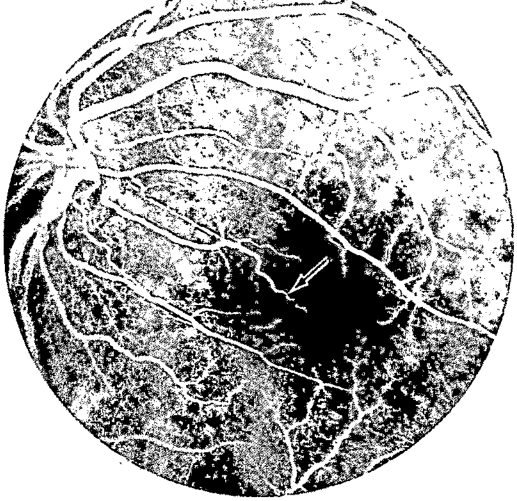

Fig. 4 (Yoshizumi, Thomas, and Hirose). Fluorescein angiogram of the macular region shows an inferiorly displaced macula with normal macular hypofluorescence. Note cilioretinal vessels passing into macular zone of hypofluorescence (arrow).

illuminance of 8.32 mL on a Goldmann perimeter, showed extensive field shrinkage. The 64-mm^2 field is smaller than our standard 1-mm^2 field. No blind spot could be plotted because of fixation difficulty caused by nystagmus. Tests of the flicker fields in both eyes, in the horizontal and vertical meridians, revealed about a 30% reduction in the critical flicker frequencies in the central and peripheral fields to 60 degrees (Fig. 5). Dark adaptation and color vision tests were within normal limits.

DISCUSSION

Curran and Robb[2] reported foveal hypoplasia in otherwise normal eyes. These eyes share several factors in common: (1) absent or indistinct foveal reflex and a uniform appearance of the macula; (2) poor central visual acuity; and (3) high association with nystagmus of the pendular or searching type.[4]

In our patient, no clear foveal reflex was observed in the macula. She had visual acuity of 6/60 (20/200) in both eyes, and a pendular, searching-type of nystagmus. These signs and symptoms have remained stationary for the past ten years.

She displayed no characteristics of albinism, no evidence of aniridia or of coloboma formation, and no myopia. Her eyes did not appear microphthalmic.

Visual-evoked-response examination revealed delayed peaks, indicating macular dysfunction. The photopic and scotopic b-waves and the a-waves of the ERG, EOG, dark adaptation, and color vision were all within normal limits, which implied relatively good overall retinal function. However, reduced amplitudes of the photopic flicker responses in ERG suggest that retinal cone function was slightly affected.

Psychophysical testing, which is usually more sensitive than electrophysiological testing, revealed more distinct abnormalities. The peripheral field examination was constricted, indicating decreased function of the peripheral retinal pathways. The sensitivity in scotopic condition, which was measured at the center of the field, mimicked rod rather than cone function. This was attributed to paramacular rod responses enhanced by fixation difficulty and caused by pendular nystagmus. In the peripheral regions to 30 degrees there was an increased rod threshold. Flicker perimetry revealed a 30% reduction in function of the retina, indicating the presence of slight, but diffuse cone disturbances (Fig. 5). Testing of retinal function revealed severe macular dysfunction and milder dysfunction of the rest of the retina.

Our case differs from the other reported cases of foveal hypoplasia in the finding of peripheral retinal rosettes, 360 degrees bilaterally, and 2 mm posterior to the ora serrata. The discrete whitish-yellowish spheres in the retina closely resemble the group B rosettes found in the peripheral fundus described by Teng and Katzin.[3] They found these structures to be about 20 μ to 1 mm in size; all were found within 2 disk diameters of the ora serrata on the retina. The smaller ones, which appeared as opaque dots and were located more anteriorly near the ora serrata, closely resembled the nodules in our case. The larger ones, which appeared to be ring-shaped and situated more posteriorly,

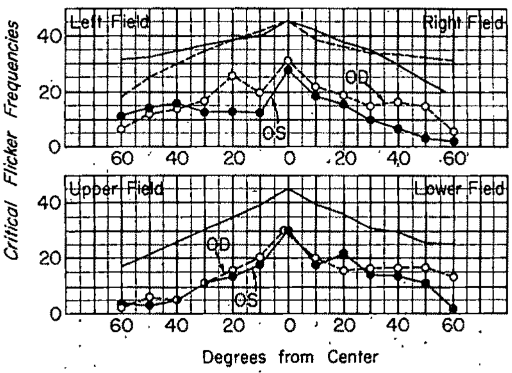

Fig. 5. (Yoshizumi, Thomas, and Hirose). Flicker perimetry in both eyes revealed about a 30% reduction in critical flicker fusion rates centrally as well as peripherally in both eyes in the vertical and horizontal meridians. In the upper graph, solid line represents right eye normal curve, broken line represents left eye normal curve. In lower graph, solid line represents normal curve for right eye and left eye.

were not present in our case. Histopathologically, these structures were found by Teng and Katzin[3] to be rosettes or neuroretinal tissue.

The rosettes in our case were distinguished from retinal pearls described by Lonn and Smith,[5] in that their position was 2 to 3 mm posterior to the ora serrata, rather than along the dentate processes, as is typical of retinal pearls. Moreover, the circumscribed 360-degree configuration of the rosette alignment was distinctly different from the typical meridional alignment of the retinal pearls along the long axis of the dentate processes.

The absence of oral bays and teeth in our case is reminiscent of the embryonic retinal ora serrata. Shortly before birth, before the 38th week of pregnancy, the meridional segmentations of the ora serrata begins to differentiate from a previously smooth peripheral retinal border.[6] However, the finding of a smooth ora serrata region alone may not be pathologic as reported by Straatsma, Foos, and Spencer[7] in a study of 200 normal eyes.

The occurrence of foveal hypoplasia, hypoplastic ora serrata, and retinal rosettes is of interest because their development may shed light on the mechanism of macular aplasia in otherwise normal eyes.

Macular and peripheral retinal differentiation occurs from the eighth month of fetal life until four months after birth.[8] The patient's mother had had whooping cough in the late third trimester of pregnancy. This may have resulted in damage to the regions of rapid differentiation in the retina, resulting in the formation of dysplastic retinal rosettes, arrested development of the ora serrata, and abnormal development of the macula.

The absence of a family history of nystagmus or decreased visual acuity, and the absence of a systemic disorder such as albinism or aniridia in our case, militates against a genetic or hereditary disease.

Lindenfeld[9] described the formation of rosettes in the region of the ora serrata in the otherwise normal eyes of a seven- to eight-month-old fetus aborted by irradiation (x-ray), because the mother suffered from mitral insufficiency. These changes have been produced experimentally by Pagenstecher[10] in the retina of fetal rabbits by x-ray. Similar observations have been made by Goldstein and Wexler[11] in irradiated human embryos.

We do not imply that all cases of foveal hypoplasia would be coupled with rosette formation in the periphery. In our case, the history of an intercurrent infection during the third trimester of pregnancy was associated with 360-degree bilateral peripheral retinal rosettes, hypoplastic ora serrata, foveal hypoplasia, and pendular nystagmus in an otherwise functional eye.

SUMMARY

A 24-year-old woman had bilateral pendular nystagmus, foveal hypoplasia, 360-degree peripheral retinal rosettes, and hypoplastic ora serrata of nongenetic origin. Kinetic perimetry revealed a generalized constriction of the field. Flicker perimetry revealed general depression of the critical flicker frequencies in the entire field. A slight increase of the threshold for light sensitivity after dark adaptation was noted from 10 to 30 degrees. Haidinger brush phenomenon was abnormal. Color vision was normal. The electroretinogram revealed mild abnormalities in the photopic flicker response. The visual-evoked response, recorded by local macular stimulation, was abnormal. The relationship of foveal hypoplasia to peripheral retinal rosettes in this case may have been caused by an intercurrent infection during the late stages of embryogenesis.

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OPHTHALMIC MINIATURE

The diagnosis of cataract is, ordinarily speaking, a matter of great simplicity and certainty, it is therefore a deplorable state of affairs to read and hear of differences of opinion on a subject susceptible of absolute accuracy. For many years past an instrument has been known, a glance through which is capable of settling with accuracy the existence or nonexistence of opacities, and it does not rebound to our credit as a profession that we have not adopted the ophthalmoscope more widely, but rather trusted to the black hat and our own often imperfect eye sight.

Journal of Comparative Pathology and Therapeutics, 1896

LACK OF HYDROPHILIC LENS DISCOLORATION IN PATIENTS USING DIPIVALYL EPINEPHRINE FOR GLAUCOMA

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Staining of ocular and adnexal structures from topically applied epinephrine has been reported for over 50 years.[1] Most frequently noted have been conjunctival[1,2] and corneal[3,4] depositions, but unusual occurrences, such as staining of an ocular prosthesis, have been reported.[5]

More recently, adrenochrome staining of hydrophilic lenses has been reported.[6,7] This phenomenon all but precludes the use of topical epinephrine in patients who wear soft contact lenses continuously. We describe herein five glaucoma patients treated with dipivalyl epinephrine in connection with soft contact lens use without development of contact lens staining.

SUBJECTS AND METHODS

Five patients received 0.1% dipivalyl epinephrine for periods of 11 to 84 weeks (Table). Patients received the medication in one eye only, one to three times per day. All patients wore their lenses 24 hours daily without removal, except patient 4, who removed the lens daily and sterilized it with hydrogen peroxide.

RESULTS

Patient 3 is of special interest. Four previous soft contact lenses developed significant coloration with epinephryl borate 1% used once a day. In each instance, the staining occurred within two weeks and increased gradually, in spite of efforts to bleach or clean the lens. At four to five months, the lenses had to be replaced because the coloration was cosmetically unacceptable.

The combination of dipivalyl epinephrine topically with soft contact lenses in place did not interfere with the effects of the drug in decreasing intraocular pressure. Four of our five patients eventually stopped using dipivalyl epinephrine or the soft contact lens, thus terminating the period of their participation in the study. Patient 1 had discontinued wearing her soft contact lens. Patient 2 no longer needed antiglaucoma medication. Patient 3 was given alternative therapy. After 27 weeks, patient 4 developed a red, irritated eye, apparently in response to dipivalyl epinephrine, and the drug was stopped. Patient 5 currently continues dipivalyl epinephrine therapy. No keratopathy, corneal fluorescein staining, or adverse reaction specifically attributable to the combination of dipivalyl epinephrine and soft contact lens was noted in any of these patients.

Three types of soft contact lenses were involved in this study, two of hydroxyethyl methacrylate (Softcon, Urocon), and one of polymethyl methacrylate and vinyl pyrrolidone (Sauflon) (Table).

DISCUSSION

Dipivalyl epinephrine is an epinephrine analogue found to be effective by topical application in the treatment of chronic open-angle glaucoma.[8] The primary indication for the use of dipivalyl epinephrine has been intolerance to epinephrine.[9] Dipivalyl epinephrine is a "pro drug" and undergoes complete conversion to epinephrine within two hours in vivo. Its ability to penetrate the human cornea is 17 times greater than that of

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TABLE

FIVE SOFT CONTACT LENS WEARERS TREATED WITH 0.1% DIPIVALYL EPINEPHRINE

Patient No., Age (yrs)	Indication for Soft Contact Lens	Frequency of Use of Dipivalyl Epinephrine	Duration of Use of Dipivalyl Epinephrine (wks)	Nature of Soft Lens
1, 68	Aphakia	Three times a day	11	Warner-Lambert (Softcon)
2, 23	Keratoconus with bullous keratopathy	Once daily	12	Sauflon 85
3, 65	Aphakia	Once daily	26	Warner-Lambert (Softcon)
4, 52	Cosmetic	Twice a day	27	Urocon
5, 50	Aphakia	Once daily	84	Warner-Lambert (Softcon)

epinephrine,[10] which is in part responsible for its effectiveness in dilute concentrations.

The biochemistry of epinephrine breakdown products in the eye is not fully understood. Epinephrine is oxidized in vivo, passing through the unstable adrenochrome phase to become one of the melanins.[1,11] Adrenochrome or its melanin derivative may be responsible for the staining of the hydrophilic contact lenses.[7] Standard epinephrine drops caused staining in from two to six weeks in three previously reported cases.[6] As previously noted, patient 4 in our series experienced similar difficulties.

The two pivalyic acid groups added to form dipivalyl epinephrine presumably render the drug less subject to oxidation and perhaps alter its breakdown products, although this has not yet been demonstrated. The alteration or absence of adrenochrome and melanin products could explain the failure of dipivalyl epinephrine to stain soft contact lenses in the dosage and time period described. The relatively low concentration of the dipivalyl epinephrine solution may also contribute to its lower potential for lens discoloration.

The problem of glaucoma treatment in patients using soft contact lenses will increase as the cosmetic, aphakic, and therapeutic use of these devices expands. The standard epinephrine compounds seem to react adversely with these lenses by causing discoloration. In our experience with five patients, use of dipivalyl epinephrine for up to 84 weeks caused no such lens discoloration. An epinephrine analogue which does not stain soft contact lenses will be helpful in the treatment of glaucoma patients who must wear these devices.

SUMMARY

Five glaucoma patients used topical dipivalyl epinephrine for periods of up to 84 weeks without discoloration of their soft contact lenses. Thus, patients who need both soft contact lenses and antiglaucoma therapy with topical epinephrine may use the dipivalyl analogue without lens spoilage.

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OPHTHALMIC MINIATURE

Post-mortem or chill cataract. As Maestrini (*Archivio di Ottalmologia,* May, June, 1915) has shown, in certain young animals, the lenses becomes partially opaque after death, and this opacity disappears on raising the temperature. The opacity appears first and remains longest in the central part of the lens. Maestrini found that in some animals in whom this cataract does not appear spontaneously, it is possible to provoke it; on the other hand, it is not possible to provoke it in many species.

Casey A. Wood, editor,
The American Encyclopedia and Dictionary of Ophthalmology
Chicago, Cleveland Press, 1918

CLINICAL COMPARISON OF DIPIVALYL EPINEPHRINE AND EPINEPHRINE IN THE TREATMENT OF GLAUCOMA

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A prodrug is an agent that undergoes biotransformation to a pharmacologically active form in the body. Dipivalyl epinephrine is a prodrug of epinephrine, formed by the addition of two pivalyl side-chains to the parent molecule (Fig. 1). Dipivalyl epinephrine is 100 to 600 times as lipophilic as epinephrine,[1-3] leading to a significant increase in penetration. It penetrates the cornea approximately 17 times more easily than epinephrine.[2] Perhaps only one-tenth the dosage of dipivalyl epinephrine is required to cause a decrease in intraocular pressure comparable to that produced by epinephrine.[1,3] Furthermore, use of lower concentrations of drug should reduce the incidence of external side effects and drug intolerance.[4] We compared dipivalyl epinephrine, 0.1%, with epinephrine hydrochloride, 2%, in a double-masked study, with respect to decrease in intraocular pressure, increase of pupil size, and incidence of side effects.

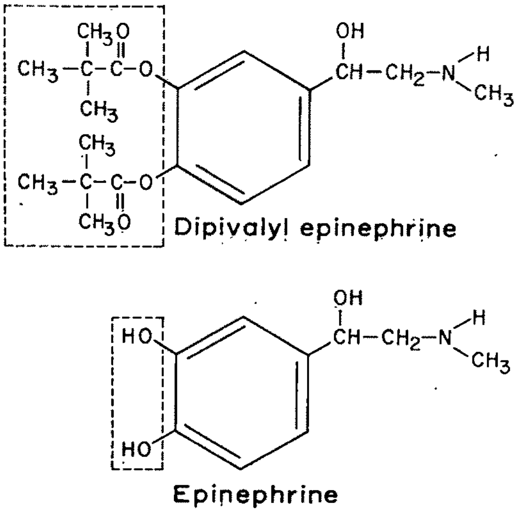

Fig. 1 (Kohn and associates). Molecular structure of dipivalyl epinephrine and epinephrine.

SUBJECTS AND METHODS

Patient population—Twenty-five patients were initially selected on the basis of having symmetric (to within 5 mm Hg) intraocular pressure $\geqq$ 22 mm Hg in each eye. Seventeen patients completed the entire six-month study and formed the statistical base for the analysis of the drugs' effectiveness. Of the eight patients who did not complete the study, three had ocular complications and five had insufficient follow-up. Seven of the 17 patients who completed the study had primary open-angle glaucoma, six had increased intraocular pressures without field loss, three had chronic angle-closure glaucoma previously treated with bilateral iridectomies, and one had open-angle glaucoma and was monocularly aphakic. There were nine women and eight men in the group. Their ages ranged from 59 to 77 years among the women and 55 to 82 years among the men, except for one 26-year-old. Three of the patients were concurrently treated with miotics or acetazolamide (Diamox), or both, during the

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Reprint requests to Steven M. Podos, M.D., Chairman, Department of Ophthalmology, Mount Sinai School of Medicine, One Gustave Levy Pl., New York, NY 10029.

entire study. Twelve of the patients had brown irides and five had either blue, green, or hazel irides.

Medications—The study was a double-masked crossover clinical trial comparing dipivalyl epinephrine, 0.1%, and epinephrine hydrochloride, 2%, over a six-month treatment period (Table 1). No epinephrine compounds were used during the initial and crossover two-week baseline periods. In the treatment periods, patients were randomly assigned one medication in a coded pink bottle for the right eye and the other medication in a coded white bottle for the left eye. Medications were taken every 12 hours and patients were examined at approximately the same time of day at least two hours after taking their drops. After the second washout period, new bottles were issued so that the eye that had received dipivalyl epinephrine now received epinephrine hydrochloride and vice versa.

RESULTS

Intraocular pressure—Seventeen patients completed the entire study, missing no more than one visit. For those patients, we found a sustained decrease in intraocular pressure with the use of dipivalyl epinephrine, 0.1%. The decrease with dipivalyl epinephrine appeared consistently less than that for epinephrine hydrochloride, 2%, but the difference between the two drugs was statistically significant ($P<.05$) only after crossover (Fig. 2, Table 2). During the first half (three months) of the study, the average intraocular pressure decreased from a baseline of 27.5 ± 0.9 mm Hg (S.E.M.) to 20.5 ± 0.8 mm Hg after three months with dipivalyl epinephrine, and from a mean baseline of 28.1 ± 0.8 mm Hg to 19.1 ± 1.2 mm Hg with epinephrine. After the crossover period, the mean intraocular pressure over the second three-month treatment period decreased from 25.9 ± 0.9 to 18.2 ± 2.1 mm Hg with dipivalyl epinephrine and from 27.2 ± 0.8 to 17.0 ± 1.9 mm Hg with epinephrine. All decreases were statistically significant ($P<.05$).

There was no statistically significant difference ($P>.05$) in response between right and left eyes treated with the same drug, nor between brown and non-brown eyes for either drug.

TABLE 1

EVALUATIONS OF STUDY PROTOCOL: PERFORMED DURING EACH VISIT

	Washout Baseline (Days)		Treatment 1 (Days)				Washout Crossover Baseline (Days)		Treatment 2 (Days)			
	−7	0	7	28	60	90	−7	0	7	28	60	90
Visual acuity	X	X	X	X	X	X	X	X	X	X	X	X
Pupil size	X	X	X	X	X	X	X	X	X	X	X	X
Applanation tonometry	X	X	X	X	X	X	X	X	X	X	X	X
Slit-lamp examination	X	X	X	X	X	X	X	X	X	X	X	X
Visual fields (Goldmann)		X				X		X				X
Tonography		X				X		X				X
Heart rate		X				X		X				X
Blood pressure		X				X		X				X
Side effects												
Ocular and systemic			X	X	X	X			X	X	X	X

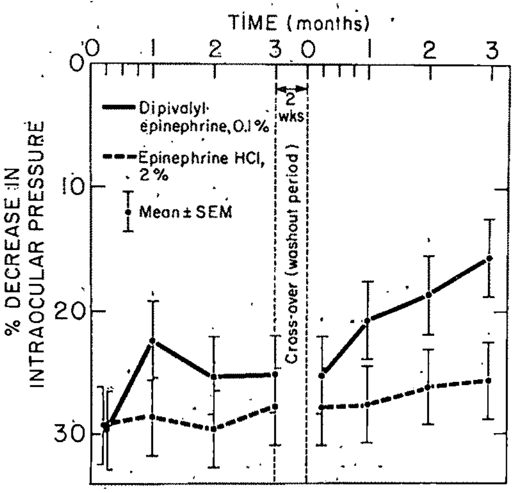

Fig. 2 (Kohn and associates). Percent decrease in intraocular pressure with dipivalyl epinephrine 0.1% vs epinephrine hydrochloride with 2% (± S.E.M. averaged over entire study).

Pupil size—The mydriatic effect of dipivalyl epinephrine, 0.1% averaged +0.65 ± 0.13 mm (S.E.M.) or +25.1%, and of epinephrine hydrochloride, 2%, +0.55 ± 0.17 mm or +18.8%, including only those patients not receiving miotics. The increases were statistically significant ($P < .05$), but there was no significant difference ($P > .05$) between the two drugs.

Ocular side effects—Three patients were discontinued from the study, two for severe epinephrine side effects, including one for classical findings of epinephrine allergy consisting of conjunctival hyperemia and follicular reaction, eyelid edema, and itching. One patient was discontinued from the study because of inadequate pressure control and progressive field loss in both eyes. Five patients did not return for sufficient follow-up.

Of the 17 patients who completed the entire study, complaints of burning occurred for eight eyes (23.5%) receiving epinephrine, and for one eye (2.9%) receiving dipivalyl epinephrine. Two patients complained of mild hyperemia on two occasions and one of blurry vision in the eye receiving dipivalyl epinephrine, but neither complaint could be confirmed on clinical examination.

Five eyes receiving dipivalyl epinephrine and four fellow eyes receiving epinephrine showed mild, inferior punctate corneal staining, each on only one occasion. One patient had bilateral involvement and four patients had involvement either only in the eye receiving dipivalyl epinephrine (one case) or at least somewhat more marked in the eye receiving dipivalyl epinephrine (three cases) than that receiving epinephrine.

Duration of effect—Residual drug effect was present between one and two weeks after cessation of both dipivalyl epinephrine and epinephrine during the crossover period. The mean increase in intraocular pressure two weeks after the final treatment day was +6.40 ± 3.9 (S.D.) mm Hg for the eye treated with dipivalyl epinephrine and +5.91 ± 3.8 mm Hg for the eye treated with epinephrine. Between the first and second week, the pressure increased +3.1 ± 4.4 mm Hg for the eye that had been treated with dipivalyl epinephrine and +2.9 ± 3.8 mm Hg for the eye that had received epinephrine. The increases over each week were statistically significant ($P < .01$), but the difference between dipivalyl epinephrine and epinephrine was insignificant (*t*-test).

Systemic side effects—A comparison of the average vital signs of patients during the treatment period with the vital signs in the second week of the two washout periods showed no significant effect on blood pressure or pulse rate. Mean blood pressure (mm Hg) when the patients were not receiving dipivalyl epinephrine or epinephrine was 140.4 ± 2.8/85.4 ± 1.5 (S.E.M.), and during the two treatment periods the mean blood pressure was 139.4 ± 2.9/85.0 ± 1.6. The mean pulse rate during the washout period was 73.1

TABLE 2

MEAN INTRAOCULAR PRESSURE (MM HG ± S.E.M.)

	Treatment 1					Treatment 2				
	Baseline	1 wk	4 wks	2 mos	3 mos	Crossover Baseline	1 wk	4 wks	2 mos	3 mos
Dipivalyl epinephrine, 0.1%	27.5 ± 0.9	19.5 ± 0.9	20.9 ± 0.6	20.4 ± 0.8	20.5 ± 0.8	25.9 ± 0.9	19.3 ± 0.8	20.4 ± 0.8	19.2 ± 1.3	18.2 ± 2.1
Epinephrine, 2%	28.1 ± 0.8	20.1 ± 1.0	19.6 ± 0.5	19.7 ± 0.7	19.1 ± 1.2	27.2 ± 0.8	19.5 ± 0.8	18.4 ± 1.2	17.2 ± 1.5	17.0 ± 1.9
No. of eyes	17	15	16	17	17	17	16	17	16	16

beats per minute ± 2.2 (S.E.M.), and during the treatment periods it was 73.6 beats per minute ± 1.8. One patient was noted to have an irregular pulse on one treatment visit only.

DISCUSSION

Dipivalyl epinephrine, 0.1%, is effective in decreasing intraocular pressure,[5-7] without causing the frequent side effects associated with epinephrine usage.[5-8] The decrease in intraocular pressure is slightly less with dipivalyl epinephrine, 0.1%, than with epinephrine hydrochloride 2%. This may occur because the ocular penetration of dipivalyl epinephrine is 17 times that of epinephrine,[2] but the concentration of dipivalyl epinephrine used was only $^1/_{20}$ that of epinephrine. The apparent further decrease in effectiveness of dipivalyl epinephrine after the crossover period requires explanation. Dipivalyl epinephrine may lose some efficacy with prolonged use, although such an effect has not been previously reported. Indeed, McClure[4] found no decrease in efficacy of dipivalyl epinephrine for periods as long as seven months. The decreased effectiveness of dipivalyl epinephrine in eyes treated with epinephrine before crossover may also result from decreased density of available β-adrenergic receptors caused by epinephrine pretreatment. Reduction of receptors has been demonstrated experimentally by Candia and Neufeld[9] in rabbit corneas after long-term epinephrine therapy.

Bischoff[5] found similar effectiveness of dipivalyl epinephrine, 0.1%, and epinephrine borate 1% in 33 patients over a three-month trial. Kriegelstein and Leydhecker[6] found no difference in the response of 16 patients to dipivalyl epinephrine 0.1% vs epinephrine hydrochloride 1%. They did find a significantly lower response with dipivalyl epinephrine 0.025%, and a greater response with dipivalyl epinephrine 0.25%, as compared to epinephrine hydrochloride 1%. Dipivalyl epinephrine 0.1% is at least equal in ability to decrease intraocular pressure as epinephrine hydrochloride 1% or borate 1%, but is somewhat less effective than epinephrine hydrochloride 2%. Theoretically, dipivalyl epinephrine 0.1% should be as effective as an intermediate epinephrine concentration, such as 1.7%.

The mydriatic effect of dipivalyl epinephrine 0.1% appears to be comparable to that of epinephrine hydrochloride 2%. Kaback and associates[7] have shown dipivalyl epinephrine 0.025% to cause no mydriasis, dipivalyl epinephrine 0.1% to cause an increase of from 0.7 to 1.6 mm over one to four hours, and dipivalyl epinephrine 0.5% to cause an increase of from 2.0 to 4.3 mm over one to four hours.

The incidence of side effects with dipivalyl epinephrine should be significantly less than with epinephrine, since the less active drug is available topically. Indeed, severe intolerance or allergy (two patients) and burning discomfort (24% of eyes) were almost uniquely associated with epinephrine usage in our study. Burning discomfort with dipivaly epinephrine occurred in only 3% of eyes. Yablonski and associates[8] studied 12 patients who had histories of epinephrine intolerance and found 83% of eyes developed allergic signs, blepharitis, or discomfort in the eye receiving epinephrine hydrochloride 2%, whereas only one eye (8%) developed allergic symptoms and signs with dipivalyl epinephrine 0.1% over an average 100-day follow-up. Kaback and associates[7] also found no particular ocular or systemic side effects with dipivalyl epinephrine 0.1%. One-tenth to $^1/_{20}$ the amount of epinephrine on the eye should reduce not only ocular discomfort, but also other side effects, such as adrenochrome deposits and cardiovascular complications.

SUMMARY

Dipivalyl epinephrine, 0.1%, though slightly less effective in decreasing intraocular pressure, showed significantly fewer side effects than epinephrine hydrochloride, 2%. Seventeen patients with symmetrically increased intraocular pressures who completed a six-month double-masked crossover study showed a significant decrease in intraocular pressure averaging 23.7% for dipivalyl epinephrine over the entire study and 27.4% for epinephrine. In the first treatment period, dipivalyl epinephrine was slightly less effective than epinephrine. In the second treatment period, dipivalyl epinephrine was statistically less effective than epinephrine. Two of the original 25 patients were dropped from the study because of epinephrine allergy or intolerance, one had uncontrolled pressures with either drug, and five failed to maintain adequate follow-up. Complaints of side effects such as burning and irritation occurred much more frequently in eyes receiving epinephrine (24%) than dipivalyl epinephrine (3%). Mild mydriasis occurred with each drug, averaging +0.65 mm with dipivalyl epinephrine and +0.55 mm Hg with epinephrine. No effect on blood pressure or pulse rate was found for the two drugs.

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IDOXURIDINE AND BACTERIAL CORNEAL INFECTION

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Idoxuridine is an antiviral agent used in the treatment of herpetic keratitis. It is also myelosuppressive[1] and retards the healing of stromal corneal wounds, presumably by inhibiting the mitosis of keratocytes.[2] In the treatment of herpetic keratitis, the advantages of idoxuridine's antiviral activity outweigh the disadvantages. Its local use is potentially immunosuppressive, however, and this and the presumed antimitotic effect could be harmful.

To test the hypothesis that idoxuridine can worsen a nonviral infection, we designed a project to determine the drug's effect on staphylococcic corneal ulcers.

MATERIAL AND METHODS

The experimental animals were 20 New Zealand white adult rabbits of both sexes, each weighing between 2.3 and 2.7 kg. Before the experiment, all of the rabbits' corneas were examined and found to be normal. The animals were divided into two groups of ten animals each. Rabbits 1 through 10 were given idoxuridine (Stoxil) ointment in both eyes four times a day for five days, and rabbits 11 through 20 were given a bland ointment base (Lacrilube) in both eyes four times a day for five days. On the sixth day the corneas of all 20 rabbits were inoculated with *Staphylococcus aureus* by the Cignetti technique,[3] and on the next three days, idoxuridine and Lacrilube were again applied.

On days 1, 2, 3, and 4 after the *S. aureus* inoculation, the corneal lesions were graded as follows: 0, no lesions or healed lesions; 1+, one small, dot-like lesion; 2+, two or more lesions, or one extensive lesion affecting less than 10% of the cornea (Fig. 1); 3+, lesions affecting 10 to 20% of the cornea; and 4+, lesions affecting more than 20% of the cornea (Fig. 2).

On postinoculation day 4 the rabbits were killed. The corneas were excised, ground, and suspended in nutrient broth. The suspension of each cornea was streaked on blood agar plates by the method described by Okumoto and associates,[4] and the plates were incubated at 37°C for 48 hours. The colonies on each plate were then counted and the counts recorded.

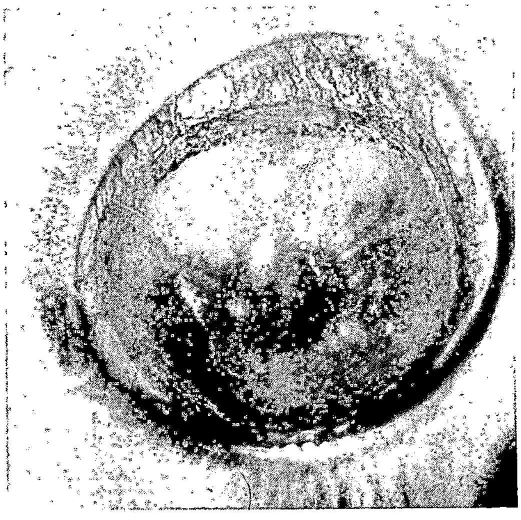

Fig. 1 (Yamaguchi and associates). Experimental staphylococcic keratitis in rabbits, grade 2+ lesion.

From the Francis I. Proctor Foundation for Research in Ophthalmology, University of California, San Francisco, California. This study was supported in part by Grants EY-01121, EY-00094, and EY-01597 from the National Institutes of Health, Bethesda, Maryland, and in part by an unrestricted grant from Research to Prevent Blindness, Inc., New York City.

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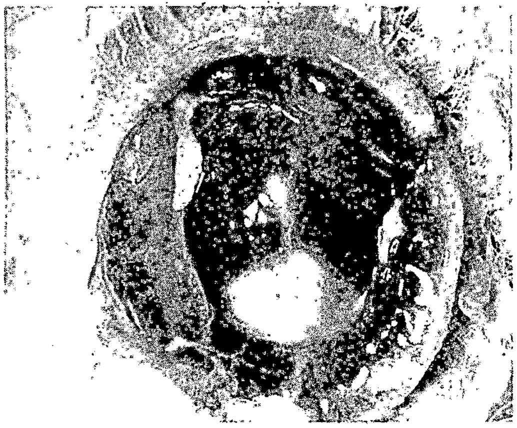

Fig. 2 (Yamaguchi and associates). Experimental staphylococcic keratitis in rabbits, grade 4+ lesion.

RESULTS

The eyes treated with idoxuridine had significantly more severe keratitis than the control group. The number of eyes with positive scores was compared with the number of negative scores (Tables 1 and 2). The number of eyes with lesions was consistently and significantly greater in Group 1 (idoxuridine-treated) than in Group 2 (Lacrilube-treated) ($P = <.05$ by Fisher's exact test).

The numbers of colonies of *S. aureus* recovered from the excised corneas were as follows: a total of 980 colonies from all eyes treated with idoxuridine, and a total of 477 colonies from all eyes treated with the bland ointment (Table 3). This too was a significant difference ($P = <.0001$ by the Student's *t*-test run on logs of counts).

DISCUSSION

Staphylococcus aureus was selected as the test bacterium because the keratitis it induces is mild and heals spontaneously in four to five days. A detrimental effect of idoxuridine would be easily detected by

TABLE 1

CLINICAL SCORES OF EXPERIMENTAL *S. AUREUS* KERATITIS TREATED WITH IDU AND LACRILUBE*

Rabbit	Day 1		Day 2		Day 3		Day 4	
No.	RE	LE	RE	LE	RE	LE	RE	LE
			Rabbits Treated with IDU					
1	1	1	2	4	2	4	1	4
2	0	1	1	2	0	1	0	1
3	3	1	3	1	2	0	2	0
4	4	1	1	1	0	0	1	1
5	1	2	1	2	1	2	1	2
6	1	2	3	2	1	4	1	4
7	1	1	1	2	1	0	1	1
8	3	0	3	1	3	0	3	0
9	0	0	1	1	0	0	0	0
10	0	1	2	1	0	1	0	1
			Rabbits Treated with Lacrilube					
11	0	0	0	1	0	0	0	0
12	1	1	2	2	0	0	1	1
13	1	0	2	0	0	0	0	0
14	0	0	0	1	0	0	0	0
15	0	1	1	0	3	3	3	3
16	1	1	1	1	1	0	1	0
17	0	1	0	1	0	0	0	0
18	1	0	1	1	2	0	2	0
19	0	0	1	1	0	0	0	0
20	0	0	1	1	0	0	0	0

*IDU signifies idoxuridine.

TABLE 2

NUMBER OF EYES WITH LESIONS VS NUMBER OF EYES WITHOUT LESIONS IN EACH GROUP OF RABBITS ON EACH POSTINOCULATION DAY*

	Day 1		Day 2		Day 3		Day 4	
Eyes†	+	−	+	−	+	−	+	−
Group 1 (IDU-treated)	15	5	20	0	11	9	14	6
Group 2 (Lacrilube-treated)	8	12	15	5	4	16	6	14

* $P < .05$ by Fisher's exact test.

†+ signifies total number of eyes with lesions; − signifies total number of eyes without lesions.

an increase in the severity of the keratitis. *Staphylococcus aureus* was also chosen because it is commonly a component of the eyelid and conjunctival flora in man. If idoxuridine did increase the susceptibility of rabbit corneas to *S. aureus* infection, it might also increase the susceptibility of human corneas.

TABLE 3

NUMBER OF COLONIES OF *S. AUREUS* RECOVERED FROM EXPERIMENTAL STAPHYLOCOCCIC KERATITIS TREATED WITH IDU AND LACRILUBE

Rabbit No.	No. of Colonies	
	RE	LE
	Rabbits Treated with IDU	
1	25	1,000 (Counted as 300)
2	0	0
3	142	24
4	5	46
5	48	65
6	126	122
7	8	5
8	48	0
9	0	8
10	3	5
	Rabbits Treated with Lacrilube	
11	0	3
12	4	2
13	0	16
14	0	102
15	5	32
16	2	0
17	0	2
18	36	246
19	6	0
20	5	16

Idoxuridine was given for five days before the bacterial inoculation (day 6), in order to immunosuppress the cornea and increase its susceptibility to bacterial infection. Idoxuridine was then continued for three more days to induce further immunosuppression. This nine-day course of idoxuridine in our experimental model simulated the treatment of humans who are often given idoxuridine for seven to ten days to heal an epithelial keratitis.

Idoxuridine increased the susceptibility of the rabbit cornea to *S. aureus* infection. On day 1, 15 eyes in the idoxuridine-treated group had lesions, compared with eight in the control group. On day 4, six eyes showed no lesions or healed lesions in the idoxuridine group, compared with 14 in the control group. Fewer eyes in the idoxuridine-treated group had healed spontaneously, and the organisms had proliferated in significantly greater numbers in the idoxuridine-treated corneas than in the untreated corneas.

We believe idoxuridine increased the susceptibility of the cornea to bacterial infection by inhibiting the local immune response. When given systemically, idoxuridine is myelosuppressive, and when given locally it may be locally immunosuppressive as well. A depression in the host's immune response would allow greater numbers of bacteria to proliferate and to cause a more severe bacterial keratitis. We observed both of these phenomena in our experiment. Idoxuridine also inhibits keratocyte regeneration,[2] and this could worsen a stromal bacterial keratitis. Idoxuridine may have similar effects on the human cornea.

If idoxuridine increases the susceptibility of the human cornea to bacterial infection, then corneas with epithelial defects that are receiving idoxuridine should be observed carefully for bacterial superinfection. The immunosuppressive effects of idoxuridine may also have an additive effect on other factors that compromise host tissue, such as age, malnutrition, corticosteroids, and disease (for example, prolonged epithelial defects, blepharitis, and keratitis sicca). Because such immunosuppressed individuals are more likely to develop secondary bacterial infection, the eyelids and conjunctivae of idoxuridine-treated patients should be cultured and all pathogens eliminated.

Because idoxuridine may be locally immunosuppressive, it should be used only when there is evidence that herpes simplex virus is the cause of the corneal disease. Occasionally, when an apparent bacterial ulcer fails to heal, herpes simplex virus is suspected, and idoxuridine is added to the antibiotic regimen. If the ulcer is indeed bacterial, the idoxuridine may worsen its clinical course.

SUMMARY

Corneas of 20 rabbits were treated with idoxuridine or a bland ointment before and after their inoculation with *Staphylococcus aureus*. The rabbit corneas treated with idoxuridine had a significantly more severe keratitis and yielded significantly greater numbers of *S. aureus* on culture than the rabbit corneas treated with the bland ointment.

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PRIMARY RADIATION THERAPY FOR MEIBOMIAN GLAND CARCINOMA

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Carcinomas of the Meibomian and other pilosebaceous glands of the eyelids are uncommon but lethal tumors. Boniuk and Zimmerman[1] reported a five-year tumor mortality of 30% and an incidence of metastasis of 28% in a series of 88 cases. Generally accepted primary treatment consists of initial wide local excision with exenteration being reserved for those patients with orbital involvement. Radiation treatment has principally been advocated only for recurrent lesions, and has been used once as primary therapy.[2]

We report herein three cases that support irradiation as the initial treatment of these tumors in selected patients.

MATERIAL AND METHODS

Histopathologic material was obtained by multiple wedge biopsy or exenteration. Serial sections were examined so that multiple tumor foci could be clearly delineated. Sections were routinely prepared following formalin fixation, paraffin embedding, and hematoxylin and eosin staining.

CASE REPORTS

Case 1—A 73-year-old man was referred to us in April 1977, with a one-year history of continuous infection of the left eye with itching, epiphora, and photophobia. He had undergone intraocular lens implantation in the right eye in January 1976. A pterygium was removed from the left eye in February 1976 and a chalazion was also removed from the left upper eyelid in April 1974. On initial examination best corrected visual acuity was R.E.: 6/24 (20/80); L.E.: counting fingers at three feet. Intraocular pressures were normal. The left eye had diffusely thickened eyelids with telangiectasia and seborrheic hyperplasia of the Meibomian glands. The left cornea had a 360-degree pannus with an eroded epithelium. The tear meniscus was greatly reduced in size in the left eye, and the lens was moderately cataractous. The left fundus was normal. Multiple wedge biopsies confirmed the diagnosis of primary multicentric Meibomian gland carcinoma in both the upper and lower left eyelids. The patient had a history of multiple myocardial infarction and was considered to be a poor risk for general anesthesia (Fig. 1, 1a and b). He was, therefore, treated with a total tumor dose of 6,600 rads over a period of 35 days in June and July 1977, by using a cobalt source and protective shields beneath the eyelids. The patient was clinically free of disease and comfortable until his death from a myocardial infarction ten months after treatment.

Case 2—A 65-year-old woman was referred to us in November 1974, with a two-year history of an irritating lesion of the right upper eyelid. Examination of biopsy specimens done by the referring physician confirmed the diagnosis of primary multicentric Meibomian carcinoma in both eyes. On initial examination, best corrected visual acuity was R.E.: 6/30 (20/100) and L.E.: 6/6 (20/20). Intraocular pressures were normal in each eye. The temporal portion of the right upper eyelid was thickened, nodular, and erythematous with multiple pustules and trichiasis. The right cornea was edematous with temporal vascularization, and the right lens was densely cataractous (Fig. 1, 2a and b). Because of the multicentric origin of the tumor, and refusal of surgery by the patient, radiation was chosen as initial therapy in this case. The patient received a total tumor dose of 4,800 rads administered over a 22-day period in November and December 1974. The globe was protected by lead shields beneath the eyelids. She did well and was apparently free of disease until May 1975, when she had a tender swelling in the right parotid region measuring 1.5 cm. In that same month, she had a right superficial

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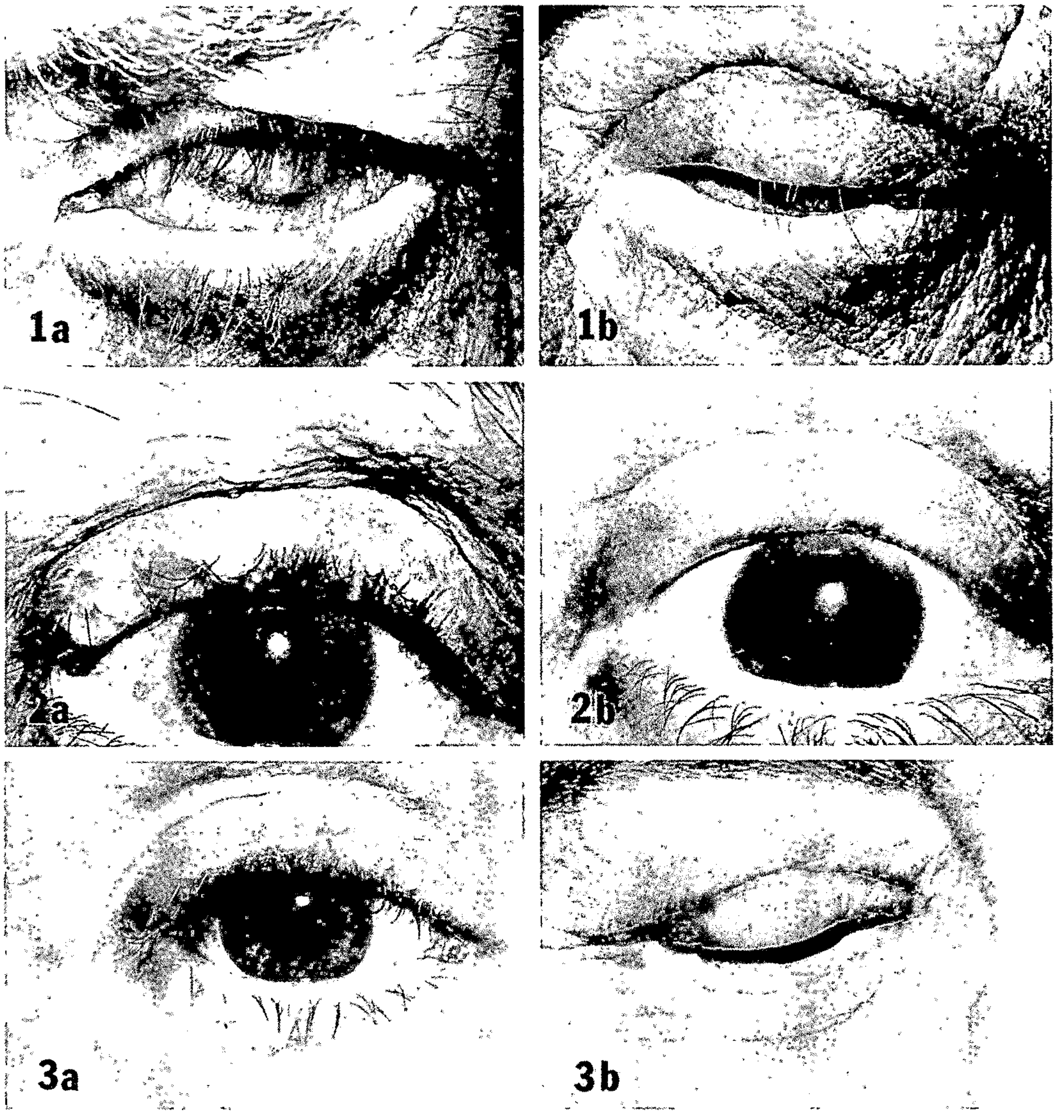

Fig. 1 (Hendley and associates). 1a, Case 1 before treatment; 1b, one month after treatment. 2a, Case 2 before treatment; 2b, three months after treatment; 3a, Case 3 before treatment; 3b, five months after treatment.

parotidectomy. A frozen section revealed Meibomian gland carcinoma, and a right radical neck disection was done at the same time. She again did well until January 1977, when yellow patches on the right upper and lower eyelids associated with renewed inflammation occurred. There was also early invasion of the right cornea by a vascular pannus. In February 1977, the patient underwent exenteration of the right orbit. The enucleated specimen showed pagetoid invasion of the corneal epithelium by the tumor. She has had no evidence of recurrence 15 months after enucleation.

Case 3—A 53-year-old woman had a three-year history of severe blepharoconjunctivitis of the right eye. This had been treated with many topical medications including corticosteroids and idoxuridine. She was first examined in August 1975. The right upper and lower eyelids were indurated and erythematous but had no loss of eyelashes. The Meibomian orifices were puckered. The conjunctiva in the right eye was injected and edematous with no papillae or follicles. The right cornea had diffuse punctate epithelial staining defects and manifested decreased sensation. The left eye was normal. A 30-day course of systemic tetracycline did not improve her blepharitis. Full-thickness wedge biopsy specimens revealed Meibomian gland carcinoma of the right upper and lower eyelids with pagetoid invasion of the overlying skin and conjunctival epithelium (Fig. 1, 3a and b). Because of the extent of the tumor, radiation was used for the initial treatment. A total tumor dose of 5,000 rads was administered to both right eyelids in March 1976 over a 23-day period. A lead contact lens shielded the globe. The patient has been free of recurrence 25 months later.

RESULTS

Multicentric tumor foci were found in both eyelids in all three cases. These were not connected on serial sections. Case 2 also demonstrated pagetoid invasion of the corneal epithelium (Fig. 2, Ib). To our knowledge, this has not been reported previously. Typical vacuolated sebaceous cells characterized this tumor (Fig. 2, Ic, d).

DISCUSSION

Only one case has been reported to our knowledge of Meibomian gland carcinoma treated initally with radiation.[2] A recurrence necessitated a second course of radiation treatment, and it was suspected that the original dose was inadequate. The patient was reported to be clinically free of disease 21 months after the second treatment.

Because of the indolent nature of Meibomian gland carcinoma, regional lymph node involvement is not positive evi-

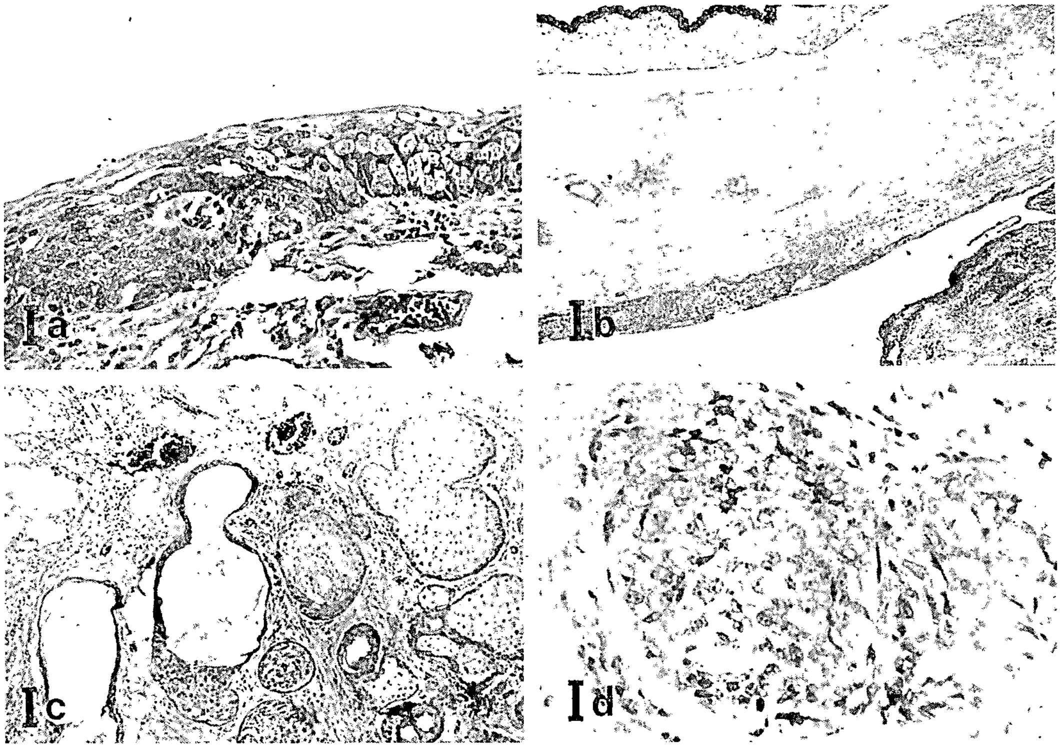

Fig. 2 (Hendley and associates). Ia, Case 2. Carcinoma in sites on surface of the upper eyelid (hematoxylin and eosin ×150); Ib, pagetoid invasion of the corneal epithelium (hematoxylin and eosin ×120). Ic, Case 1. Appearance of tumor in upper eyelid (hematoxylin and eosin ×100); Id, higher power appearance of tumor in upper eyelid (hematoxylin and eosin ×200).

dence of systemic metastasis. One of our patients has remained free of metastatic disease following a lymph node dissection despite a recurrence at the primary site. A patient with multiple sebaceous tumors may be at greater risk for malignancies of internal organs. The existence of this rare syndrome has been postulated by Bakker, Tjon, and Joe.[3]

Since Meibomain gland carcinoma can be multicentric,[4] complete surgical removal may be difficult, and the general anesthesia frequently required may be an unacceptable risk with some patients. Radiation damage to the anterior segment of the globe is always possible, but this can be minimized by the use of radiation shields beneath the eyelids, as was done in our three patients. We believe the evidence warrants further investigation of radiation therapy as initial treatment for sebaceous gland carcinoma of the eyelids in selected individuals, particularly in elderly patients who refuse initial exenteration or those who are unacceptable risks for anesthesia.

SUMMARY

Three patients with Meibomian gland carcinoma were treated initially with irradiation. This therapy was palliative in one case that eventually required orbital exenteration because of recurrent disease. A second patient was clinically tumor-free ten months after treatment when he died of a myocardial infarction. The third patient has shown no evidence of recurrent disease in the 25 months since treatment.

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PATIENT COMPLIANCE WITH USE OF TOPICAL OPHTHALMIC CORTICOSTEROID SUSPENSIONS

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Improper use of medication by patients is an important factor in the irregular response of disease to treatment. Compliance depends to a large extent on the patient's understanding of the nature of the medication prescribed and its use, as well as on the complexity of the treatment regimen.[1-5]

When topical ophthalmic corticosteroid suspensions are prescribed for the treatment of ocular inflammation, the patient must follow the dosage schedule and shake the bottle to suspend the drug fully so the full dosage of medication is delivered to the eye. We wondered if the irregular response of ocular inflammation to topical corticosteroid suspensions in some of our patients might be related to inadequate dosage, resulting from not shaking the bottle to suspend the corticosteroid fully.

We report herein a study of patient compliance with the use of topical corticosteroid suspensions and an analysis of the concentration of corticosteroid delivered when the drug bottles are shaken a number of times or not at all.

SUBJECTS AND METHODS

The study included 100 adult patients. Each subject was given a bottle of 1.0% prednisolone acetate suspension labeled, "Instill one drop in each eye four times daily. Shake well." The label was typed in black letters except for the instruction to shake well, which was in red for emphasis. All patients could read the label easily. The subjects were shown a manikin head placed face up and told, "Pretend these eye drops were prescribed for a relative or friend, represented by this head, and you are to put the drops in his (or her) eyes. Please read the instructions carefully, then show us how you would put the drops in the eyes (of the manikin)." A manikin head was used to avoid anxiety that might arise in a patient at the thought of putting drops in his own eyes. The request was presented so that the importance of reading the instructions carefully and the manner of instillation of the drops were not overemphasized. We observed the number of times the bottle was shaken. One back-and-forth motion was considered one shake.

To determine the concentration of corticosteroid delivered after a number of shakes, 5-ml bottles of four commonly available ophthalmic corticosteroid suspensions were left upright and undisturbed for two weeks to allow the drug to settle. The preparations used were two brands of 1.0% prednisolone acetate (Pred Forte and Econopred Plus), 0.1% dexamethasone (Maxidex), and 2.5% hydrocortisone acetate (Hydrocortone). Pred Forte and Econopred Plus were in opaque plastic bottles, Maxidex in a clear plastic bottle, and Hydrocortone in a brown glass bottle. Samples were collected from separate, unused bottles of each preparation after 0, 5, 10, 15, 20, 25, 30, and 40 shakes. Each bottle was shaken by hand in a uniform manner by inverting the bottle smoothly to effect a back-and-forth movement through 180 degrees. We thought this was a reasonable technique.

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because the subjects varied in the way they shook the bottle. To collect each sample, five drops were squeezed onto a sheet of Parafilm and 100 μl was pipetted into a vial. The corticosteroid concentration of each sample was measured by using a modified isonicotinic acid hydrazide assay[6] that tests for the A ring of corticosteroids. We collected and analyzed six sets of samples. The mean of the six concentrations obtained for each number of shakes was used to analyze the results. To obtain the maximum concentration of drug attainable for the preparations tested, two bottles of each were shaken on a mechanical rotator for 20 minutes. One bottle of each drug was removed from the rotator, and two samples were collected and analyzed. Additional shaking of the other bottles on the rotator did not yield a significantly different concentration of drug. The mean concentration of samples obtained after 20 minutes of shaking was used as the maximum attainable concentration.

RESULTS

Of the 100 patients in our study, 63 did not shake the bottle after reading the label. Of the remaining 37 patients, six shook the bottle one to five times; 12, six to ten times; ten, 11 to 15 times; six, 16 to 20 times; two, 21 to 25 times; and one patient shook the bottle 32 times (Table 1).

TABLE 1

PATIENT COMPLIANCE IN SHAKING BOTTLE OF CORTICOSTEROID AFTER READING INSTRUCTIONS

No. of Shakes	No. of Subjects (Total, 100)
0	63
1-5	6
6-10	12
11-15	10
16-20	6
21-25	2
26-30	0
31-35	1
36-40	0

Figure 1 shows the mean concentration of corticosteroid obtained after shaking fresh bottles of each of the four drugs 0, 5, 10, 15, 20, 25, 30, and 40 times. The maximum concentration obtained after shaking each preparation on the mechanical rotator is also indicated. Table 2 and Figure 2 show the percentage of maximum concentration of each drug obtained after shaking the bottle in increments of five times. Pred Forte, Maxidex, and Hydrocortone attained 53, 43, and 41%, respectively, of their maximum concentrations after ten shakes; Econopred Plus attained 14% of its maximum concentration. After 15 shakes, Pred Forte, Maxidex, and Hydrocortone reached 61, 43, and 58%, respectively, of their maximum concentrations, while Econopred Plus reached 9%. After 40 shakes, Pred Forte, Maxidex, and Hydrocortone attained 82,

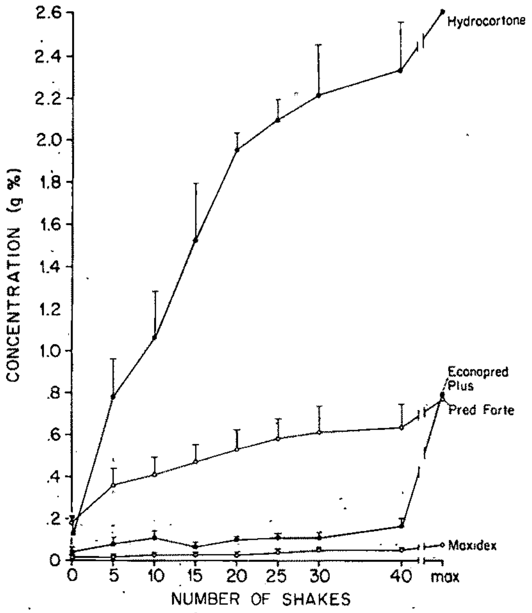

Fig. 1. (Apt, Henrick, and Silverman). Corticosteroid concentration in relation to number of times bottle is shaken. Represents mean of six concentrations plus standard error of the mean.

TABLE 2

MEAN PERCENTAGE OF MAXIMUM CONCENTRATION AFTER SHAKING BOTTLE

No. of Shakes	Pred Forte	Econopred Plus	Maxidex	Hydrocortone
0	23	5	29	5
5	47	10	29	30
10	53	14	43	41
15	61	9	43	58
20	69	13	43	75
25	75	14	57	80
30	79	14	71	85
40	82	22	71	90

71, and 90%, respectively, of their maximum concentrations; Econopred Plus attained 22% of maximum. The data were analyzed statistically by using the Student *t*-test, and all the concentrations indicated in Figure 1, except one (the concentration of Hydrocortone after 40 shakes), were significantly less than the maximum concentration ($P<.05$).

During the sample collection Econopred Plus was more difficult to bring into suspension than the other preparations. Several of the Econopred Plus bottles had dried material plugging their tips so that no drops could be squeezed out unless the plugs were pushed in with a needle.

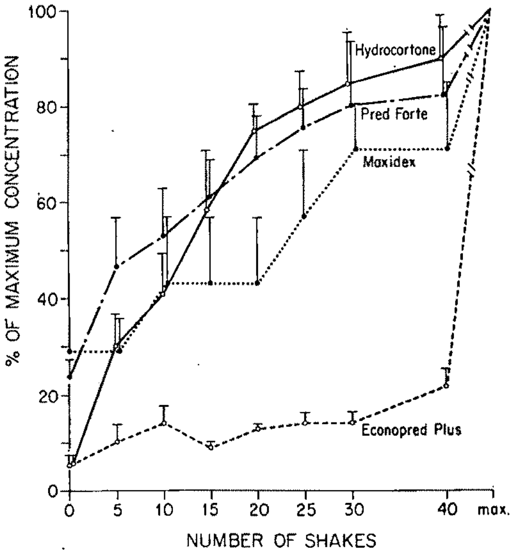

Fig. 2 (Apt, Henrick, and Silverman). Percent of maximum corticosteroid concentration in relation to number of times bottle is shaken. Represents mean of six concentrations plus standard error of the mean.

DISCUSSION

Most patients in our study (63%) did not shake the bottle at all after reading the label even though the instruction to shake well was emphasized in red letters. Of the 37 patients who did shake the bottle, 18 (48.6%) did so ten times or fewer, and 28 (75.7%) shook it 15 times or fewer. Only one patient shook the bottle as many as 32 times.

The patients in this study who did not shake the bottle would have delivered to the eye 29% or less of the maximum concentration of any of the four corticosteroid preparations tested. Almost half (48.6%) of the patients who did shake the bottle (bottle shaken one to ten times) would have delivered about half (53%) or less of the maximum concentration of any of the four drugs. Three-fourths (75.7%) of the patients who shook the bottle (bottle shaken one to 15 times) would have delivered less than two thirds (61%) or less of the maximum concentration of any of the four medications (Table 2).

Pred Forte and Hydrocortone suspended more easily than the other two corticosteroid suspensions. The percent of maximum concentration of Pred Forte and Hydrocortone varied almost linearly with the number of shakes. Even after shaking the bottle 40 times, however, Pred Forte did not reach its maximum concentration (Fig. 1). The percent of maximum concentration of Maxidex varied less linearly with the number of shakes (Fig. 2), probably because the isonicotinic acid hydrazide assay becomes less sensitive in the presence of low corticosteroid concentrations. Econopred Plus was the most difficult preparation to get into suspension; after 40 shakes, only 22% of the maximum concentration was attained. Only Hydrocortone reached its maximum concentration after 40 shakes. Each of the four preparations is formulated differently. Differences in vehicle, particle size, and other factors might account for the variance in number of shakes required to suspend each drug.

Corticosteroid suspensions, rather than solutions, are often prescribed for ocular inflammation, because they are better absorbed through the cornea.[7,8] Lack of patient compliance with shaking these suspensions and the difficulty of getting some of these drugs into suspension, as shown in this study, could result in a lower concentration of corticosteroid delivered to the site of inflammation than one might expect from the designation on the label.

Many of the corticosteroid suspensions are packaged in opaque bottles and it is not possible to see whether the drug is fully suspended. Manufacturers should consider packaging the suspensions in clear bottles to allow a visual check of the suspended corticosteroid.

Since none of our subjects, after reading the label, shook the bottle sufficiently to suspend the corticosteroid fully, we recommend that the manufacturer specify the number of times the bottle of corticosteroid should be shaken to suspend the drug fully. Additionally, the physician and the pharmacist, when dispensing the prescription and the drug, should verbally instruct the patient about the importance of shaking the bottle the necessary number of times.

The results of our study represent the amount of drug delivered to the eye on initial shaking of the bottle. With repeated use of the preparation, more drug would go into suspension, but on the basis of our compliance survey, it is likely that many patients would still not receive the maximum concentration of the drug.

SUMMARY

We surveyed the compliance of 100 patients with the instructions on the label of a bottle of 1.0% prednisolone acetate ophthalmic suspension. Of these patients, 63 did not shake the bottle, as directed on the label. Of the 37 subjects who did shake the bottle, 18 (48.6%) did so one to ten times, 28 (75.7%) shook it one to 15 times, and only one patient shook the bottle as many as 32 times.

We determined the concentration of corticosteroid in four commonly available preparations of ophthalmic corticosteroid suspension after shaking separate, unused bottles of each drug up to 40 times. The preparations tested were 1.0% prednisolone acetate (Pred Forte and Econopred Plus), 0.1% dexamethasone (Maxidex), and 2.5% hydrocortisone acetate (Hydrocortone). The percent of maximum concentration of each of these preparations was 29% or less after no shakes, 53% or less after ten shakes, and 61% or less after 15 shakes. After shaking the bottle as many as 40 times, only Hydrocortone reached its maximum attainable concentration.

Almost two thirds of the subjects in this study, those who did not shake the bottle, would have delivered to an eye less than one third of the maximum concentration of any of the medications tested. Almost half of the patients who did shake the bottle (bottle shaken one to ten times) would have delivered half or less of the maximum concentration of any of the four drugs. Three-fourths of the patients who shook the bottle (bottle shaken one to 15 times) would have delivered less than two thirds of the maximum concentration of any of the four medications.

ACKNOWLEDGMENTS

The isonicotinic acid hydrazide reagent and the prednisolone, dexamethasone, and hydrocortisone standards used in the assays were provided through the courtesy of Mr. Hampar Karageozian, Director of Pharmaceutical Services, Allergan Pharmaceuticals, Irvine, California.

Bottles of Econopred Plus and Maxidex were supplied by Alcon Laboratories, Inc., Fort Worth, Texas; Pred Forte by Allergan Pharmaceuticals,

Irvine, California; and Hydrocortone by Merck Sharpe and Dohme, West Point, Pennsylvania.

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OPHTHALMIC MINIATURE

"What symptoms may I expect?" asked Danbury.

"It generally takes the form of optical delusions."

"I see specks floating all about."

"That is mere biliousness," said the doctor soothingly, for he saw that the lad was highly strung and he did not wish to overdo it. "I dare say that you will have no symptoms of the kind, but when they do come they usually take the shape of insects, or reptiles, or curious animals."

"And if I see anything of the kind?"

"If you do, you will at once send for me;" and so, with a promise of medicine, the doctor departed.

Sir Arthur Conan Doyle
The Green Flag & Other Stories 1900

IMMUNE RESPONSIVENESS IN PATIENTS WITH CHOROIDAL MALIGNANT MELANOMA

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DOUGLAS J. PRITCHARD, M.D., AND DUANE M. ILSTRUP
Rochester, Minnesota

Although the accuracy of the clinical diagnosis of choroidal malignant melanoma has improved considerably, uncertainties still exist regarding the effectiveness of enucleation for the management of this tumor. Some investigators urge early enucleation for small melanomas, and others have offered alternatives to definitive enucleation, such as excisional biopsy, high-intensity local irradiation, photocoagulation, administration of cytotoxic agents, or simple observation as well as immunotherapy.

Baseline information is minimal on the immunologic responsiveness of patients with ocular melanoma. Our study analyzes information provided by selective serial multivariant immunologic testing of patients with choroidal malignant melanoma. An immune profile was identified by determining cellular immunologic responsiveness using standard recall antigens for skin testing, leukocyte enumerations, T- and B-cell assays, and mitogen-induced lymphocyte blastogenesis. Information on these immune responses was correlated with initial clinical status, histopathologic findings, and the development of metastasis. Additionally, comparisons were made between the group with choroidal malignant melanoma and an age-matched control group.

SUBJECTS AND METHODS

This study involved 25 patients with choroidal malignant melanoma who were seen at the Mayo Clinic from 1974 to 1977. The ages of the patients ranged from 31 to 74 years, with a median of 59 years (Fig. 1). Histopathologic findings in 23 patients were available (Fig. 2); enucleation was not performed in two patients. One patient had a presumed diagnosis of malignant melanoma made during a follow-up period of 35 years. The second patient had a primary malignant melanoma of the choroid, and enucleation was not performed because of metastasis that was diagnosed when he entered the study. Nine patients had recognized metastases four to 72 months after entrance into the study. Eight of these patients died during the investigation.

Selective immunologic tests included both delayed-hypersensitivity and cell-mediated immunity studies. These tests

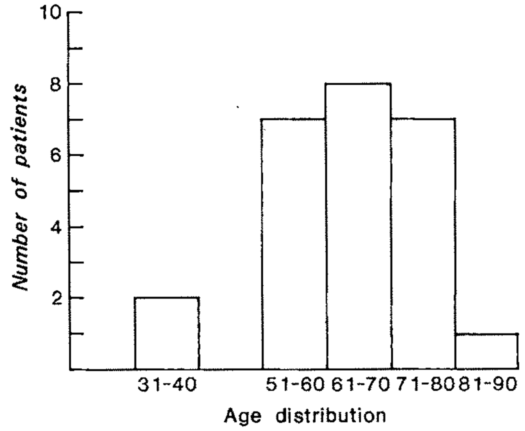

Fig. 1 (Priluck and associates). Age distribution of 25 patients with choroidal malignant melanoma.

From the Departments of Ophthalmology (Drs. Robertson and Priluck), Orthopedics and Oncology (Dr. Pritchard), and the Section of Medical Research Statistics (Mr. Ilstrup), Mayo Clinic. This study was supported in part by Research Grant CA-11911 from the National Institutes of Health, Public Health Service (Dr. Pritchard), and a research grant from Fight for Sight, Inc., New York City (Dr. Priluck).

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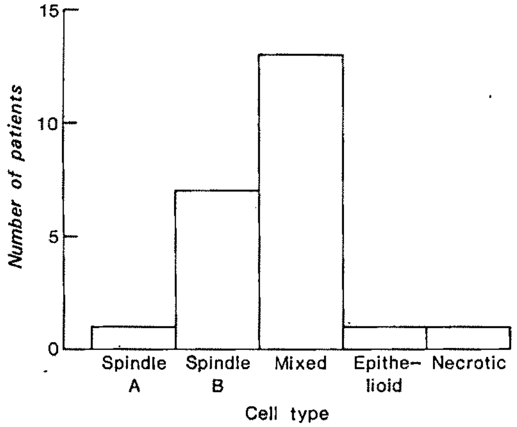

Fig. 2 (Priluck and associates). Histopathologic diagnosis in 23 eyes enucleated for choroidal malignant melanoma.

were performed in the Orthopedic Oncology Research Laboratory under the direct supervision of one of us (D. J. P.). Normal, healthy persons of similar age, who were not taking systemic medications, served as controls. The immune studies were repeated at approximately six-month intervals throughout the investigation.

Cellular immunologic responsiveness was determined by skin testing for delayed hypersensitivity with the following antigens: purified-protein derivative of tuberculin, 5 tuberculin units; streptokinase-streptodornase, 50 mg; and canadin, 0.1 ml of a 1:100 dilution of the aqueous batch preparation of *Monilia albicans*. Phosphate-buffered saline, 0.1 ml, was used as the control skin test. The results of skin tests were recorded as the average of two perpendicular diameters of induration measured at 48 hours, less the extent of induration observed at the injection site of the buffered-saline control. A positive reaction was any measurable response greater than that recorded at the control site.

Nonspecific lymphocyte blastogenesis was performed by a semimicrotechnique as reported by Webel, Briggs, and Ritts.[1] The lymphocytes were set in quadruplicate culture for each patient. Lymphocytes were respectively studied by using three mitogens: concanavallin A, 10 mg; phytohemagglutinin, 0.10 mg; and pokeweed, 10 mg. A three-day culture period was used for assessment of lymphocyte blastogenesis.

Thymus-dependent lymphocytes (T cells) and peripheral lymphocytes (B cells) were assayed by FITC-labeled anti $(Fab)_2$ human immunoglobulin, as described by Hepburn and Ritts.[2] T-cell data were analyzed by the T-cell binding of three or more sheep erythrocytes. Latex ingestion, peroxidase staining, and cytoplasmic esterase staining were used to eliminate small monocytes as part of the lymphocyte population.[3,4]

One-way analysis of variance was performed for each of the immune studies to test whether the means differed among patients with melanoma and no metastasis, patients with melanoma and metastases, and control patients.[5] If this test was significant, the least significant difference was used to determine which pairs of mean values were significantly different. Pairwise comparisons were then made between the initial results of these two respective groups and controls. Additionally, Student's *t*-tests[5] were performed for all immune factors to determine whether the average immune profile from the initial to the last evaluation changed significantly in those patients in whom metastatic disease developed during the investigation; a similar analysis was done for patients who remained free of metastasis. Patients whose data were analyzed in this way met the criteria of having a minimum of three serial immune profiles during the course of this study. Student's *t*-tests also were performed to determine whether differences in the initial immune profiles reflected differences between histologic types spindle A and B, and mixed, epithelioid, and necrotic types.

TABLE 1
INITIAL LEUKOCYTE ENUMERATIONS (M ± SD) IN PATIENTS WITH MALIGNANT MELANOMA AND IN CONTROLS

	Leukocytes		Lymphocytes		T Cell		B Cell	
	No.	(per mm^3)	No.	(per mm^3)	No.	(per mm^3)*	No.	(per mm^3)*
Choroidal melanoma, no metastasis	15	7,540 ± 2,283†	14	2,009 ± 811	9	1,406 ± 739	9	222 ± 130
Choroidal melanoma, metastasis	10	6,490 ± 1,947	10	1,365 ± 556‡	7	735 ± 372	7	171 ± 99§
Controls	51	6,200 ± 1,328	51	1,968 ± 467	51	1,088 ± 399	50	289 ± 96

*T and B cells are percentages of total lymphocytes.
Significant difference from controls:
†P<.007
‡P<.002
§P<.005.

Results

The initial mean total leukocyte count was 7,540/mm^3 for patients with choroidal malignant melanoma without metastases and 6,490/mm^3 for patients in whom metastatic disease developed during the course of this study. There was a significant increase of total leukocytes between the group without metastatic disease and the control group (6,200/mm^3) (P<.007). However, there was a significant decrease of total lymphocytes in the group with metastatic disease (1,365/mm^3) as compared with the control group (1,968/mm^3) (P<.002) (Table 1).

There was no significant difference in the initial total T-cell counts between the control group (1,088/mm^3) and patients with melanoma whether they developed metastatic disease (735/mm^3) or remained free of metastasis (1,406/mm^3). However, a significant depression of initial B-cell enumerations was noted between the group of patients in whom metastatic disease developed during the course of this study (171/mm^3) and the control group (289/mm^3) (P<.005) (Table 1).

The patients with melanoma who had evidence of metastatic disease had a significant depression of nonspecific lymphocyte blastogenesis in the presence of three mitogens (concanavallin, phytohemagglutin, and pokeweed) at the time of initial testing. Concanavallin-stimulated lymphocyte transformation also was significantly depressed in patients with choroidal malignant melanoma without metastases (Table 2). Although as a group, the patients with metastases had a significant depression of their initial mitogen-induced lymphocyte blastogenesis, individual responses frequently overlapped responses of the controls. A wide overlap existed for the individual responses for concanavallin mitogen-induced lymphocyte transformation between controls and patients with and without metastases (Fig. 3). A similar overlap existed among lymphocyte transformation studies using phytohemagglutinin and pokeweed. Lymphocyte blastogenesis did not differ in patients with various melanoma cell types, nor did serial studies of lymphocyte blastogenesis change when metastasis developed.

The mean size of delayed-type skin hypersensitivity responses using three different antigens (streptokinase-streptodornase, purified-protein derivative of tuberculin, and *Candida*) in patients with choroidal malignant melanoma did not significantly differ from the control group. This was true for both the group that was metastasis-free and the group

TABLE 2
INITIAL MITOGEN-INDUCED BLASTOGENESIS (M ± SD) IN PATIENTS WITH MALIGNANT MELANOMA AND IN CONTROLS

	No.	Concanavallin (cpm ×10³)	No.	Pokeweed (cpm ×10³)	No.	Phytohemagglutinin (cpm ×10³)
Choroidal melanoma, no metastasis	11	17 ± 14*	12	13 ± 8	12	35 ± 28
Choroidal melanoma, metastasis	9	6 ± 4†	9	7 ± 7‡	9	19 ± 24§
Controls	42	35 ± 25	49	20 ± 18	61	49 ± 32

Significant difference from controls:
*P<.02
†P<.001
‡P<.05
§P<.01.

with recurrent tumor. None of the patients studied had anergy.

Metastatic disease developed in nine patients during the study; one patient had recognized metastasis at the time he entered the study. Of this group, eight patients died of metastases within a follow-up period of three to 41 months. The median time between entrance into the study and the onset of metastasis was 21 months. Four patients with metastatic disease had sufficient serial data of their immunologic factors to allow statistical analysis. When all immune testing variables were considered, none of these four had significant alterations in the serial profiles during the course of the investigation. Although most of these patients died from metastatic disease, their respective serial immune profiles remained unaltered. Similarly, none of the patients who remained free of metastases had alteration in their serial immune profiles.

The eight patients who died had either mixed or epithelial cell types, whereas none of the patients with spindle cell types had metastatic disease during a mean observation period of 3.7 years. Nevertheless, there was no statistical alteration in initial immune profiles of the eight patients who had spindle cell types A and B when compared with the 15 patients who had mixed, epithelioid, and necrotic cell types. One patient with the clinical diagnosis of choroidal malignant melanoma who has been observed for 35

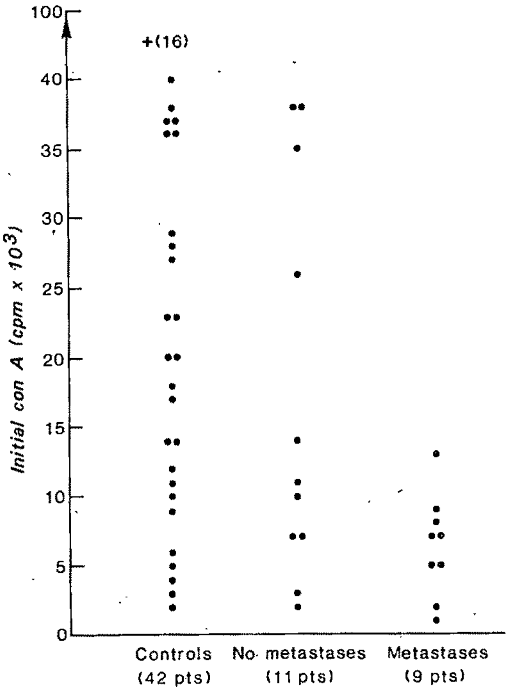

Fig. 3 (Priluck and associates). Individual responses of concanavallin mitogen-induced lymphocyte blastogenesis in patients with choroidal malignant melanoma compared with an age-matched control population. Note wide range of responses and overlap among the three groups.

years had no peculiarities of his immune profile when compared with the control group. The other patient without histopathologic confirmation, who had metastatic disease at the time he entered the study, had no apparent alterations in his immune profile, as compared with controls.

DISCUSSION

Host immunity plays a vital role in a person's capacity to cope with a wide variety of malignant neoplasms, including malignant melanoma.[6-11] Immune studies of patients with melanoma of the skin have shown alterations in both delayed hypersensitivity and cell-mediated systems.[10-12] The role of host immunity in patients with malignant skin melanoma has been supported by the observations that Bacillus Calmette Guérin used as an immunologic adjuvant, may lead to therapeutic regression[13]; that antibodies have been demonstrated against malignant melanoma by immunofluorescent techniques[14]; and that sera containing IgM and IgG of patients with skin melanoma react specifically with autochthonous and allogenic melanoma antigens.[15] Additionally, delayed-type hypersensitivity and mitogen-induced lymphocyte blastogenesis have been noted to be impaired in patients with skin melanoma.[10,11,16] Finally, the clinical observations of the capricious behavior of malignant melanoma, including spontaneous regression of skin melanoma, indicate that host immunocompetence may have an important role in the course of this tumor.

Immunocompetence may be a key factor in explaining the variable behavior of malignant melanoma of the choroid. The premise that cancer regeneration evokes immune alterations in patients with malignant melanoma of the eye has been widely investigated. Autologous tumor-associated antibodies have been found in the sera of patients with choroidal malignant melanoma by indirect immunofluorescent techniques.[17,18] Antitumor IgM antibodies have been detected in patients with uveal and conjunctival melanoma.[19] Rahi[20] found autoantibodies reactive against a patient's own melanoma cells by using complement-dependent cytotoxicity studies. Additionally, Char and associates[21] obtained a positive, cutaneous, delayed-hypersensitivity response in 18 of 19 patients with confirmed ocular melanoma when tested with a soluble melanoma antigen. Abnormal leukocyte migration inhibition, which correlated with in vivo delayed hypersensitivity,[22,23] also has been found in five of seven patients with choroidal melanoma.[24]

Specific alterations of one's immune status may indicate the development of metastatic disease in certain neoplasms or may define how a patient's host defenses will affect the natural history of the tumor. Several studies have noted depressed immunocompetence in advanced metastatic disease.[25] Others have suggested that host immunity, as evidenced by responsiveness to skin testing, can be related to the stage of certain types of carcinoma.[26] With respect to skin melanoma, accumulation of extensive data has failed to support the premise that a person's capacity to contain or retard malignancy is related to deterioration of immunologic status.[11,26,27]

SUMMARY

We studied selective in vivo and in vitro immunologic factors of 25 patients with choroidal malignant melanoma. Immune profile factors did not show consistent differences between patients with melanoma and an age-matched control population. The studies were also noncontributory in delineating variations in immunocompetence with respect to melanoma cell types. Serial immune profiles failed to identify a change when clinical evidence of metastasis developed. When

tested as a group, patients with melanoma in whom metastatic disease developed during the study showed significant depression of initial mitogen-induced lymphocyte blastogenesis. This depression was noted in advance of clinically recognized metastatic disease. This group had a wide range of responses that overlapped those of control patients, thus making the usefulness of these testing factors questionable for individual prognosis or for clinical monitoring.

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OPTIC ATROPHY IN ACUTE INTERMITTENT PORPHYRIA

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Acute intermittent porphyria is a Mendelian inherited dominant defect in porphyrin synthesis.[1,2] We report herein a case with visual loss as an unusual complication of acute intermittent porphyria.

CASE REPORT

A 24-year-old woman had her first seizure in December 1967. All seizures were nonfocal, grand mal type without an aura, with postictal drowsiness, and severe frontal headaches. She was treated as an outpatient with phenytoin sodium and phenobarbital, but satisfactory control of her seizures was never achieved.

In May 1970, she was hospitalized for possible phenytoin toxicity. Results of ophthalmologic examination were normal. Seizure activity continued intermittently despite oral medication. During 1971, she was examined in a number of hospitals; neurologic examination including electroencephalography was reported as normal. It was thought that her seizures were hysterical in nature, and she was instructed to continue both psychiatric counselling and anticonvulsant therapy.

In May 1973, her condition deteriorated and seizures occurred as frequently as once a day. On May 11, 1973, in addition to seizures, she had nausea, vomiting, and abdominal pain; she was hospitalized on May 15, 1973. At that time, the only significant finding was hypokalemia with accompanying electrocardiographic changes. She was treated with phenytoin, potassium, and intramuscular chlorpromazine. On May 18, 1973, she lost control of both bladder and bowel. No localizing neurologic signs were evident. Cerebrospinal fluid examination revealed no abnormalities; blood glucose was within normal limits. Trifluoperazine was added to the treatment. Her level of consciousness improved and by May 22, 1973, she had regained control of bladder and bowel. As she became more responsive, she complained of the inability to see anything except light. Loss of vision was diagnosed as hysterical. Pupils were noted to be responsive, however, no ophthalmologic examination was performed. She was transferred to a psychiatric hospital with the diagnosis of psychotic reaction. Treatment consisted of phenytoin, phenobarbital, chlorpromazine, trifluoperazine HCl, and trihexyphenidyl HCl. She left the psychiatric hospital one week later.

Over a three-week period, her vision gradually, but incompletely, returned. She did not see anything from her left eye. On June 4, 1973, the patient came to here for diagnosis and treatment of her seizure disorder and visual problem. On the basis of the following laboratory tests, a primary diagnosis of acute intermittent porphyria was made: a positive urinary Watson-Schwartz test, urine porphobilinogen of 32.1 mg/24 hr ($N<2$ mg/24 hr), urine Δ amino levulinic acid of 13.0 mg/24 hr ($N \leq 2.5$ mg/24 hr), and urine coproporphyrin of 55 μg/24 hr ($N=0\text{-}160$ μg/24 hr). Discontinuance of her previous chemotherapy, careful medical management with dietary consultation, and 5 mg of diazepam once daily controlled her seizures. Her right eye was normal. In the left eye visual acuity was hand movements and a Marcus Gunn pupil with a pale optic disk was noted.

Neuro-ophthalmologic examination was performed initially by us in June 1977. Visual acuity was R.E.: 6/6 (20/20), and L.E.: counting fingers at 30 cm. She identified all color plates with the right eye, but none in the left eye. A left afferent pupillary defect was present. Goldmann perimetry revealed unilateral visual loss (Fig. 1). The left optic disk was diffusely pale with marked thinning of the nerve fiber layer (Fig. 2). The remainder of the neurologic examination was unremarkable.

Radiologic evaluation of the skull, orbits, paranasal sinuses, and optic foramina was normal. An electroencephalogram and computed transaxial tomography were normal.

Pattern reversal visual-evoked responses were normal in the right eye (latency 94 msec, amplitude 6.8 mV), while there was no response to pattern reversal stimulus and only a small response to flash stimulus in the left eye (latency 108 msec, amplitude 5.9 mV). Electroretinography was normal in both eyes.

DISCUSSION

The symptoms and course of acute intermittent porphyria are extremely variable. Abdominal pain, nausea, vomiting, constipation, psychiatric disturbances, various neurologic disorders such as hypesthesia, severe pain in the extremeties, paraplegia or quadriplegia, and convulsive seizures have been reported alone or in combination as symptoms.[1-5] Attacks of acute intermittent porphyria may occur spontaneously or may be associated with

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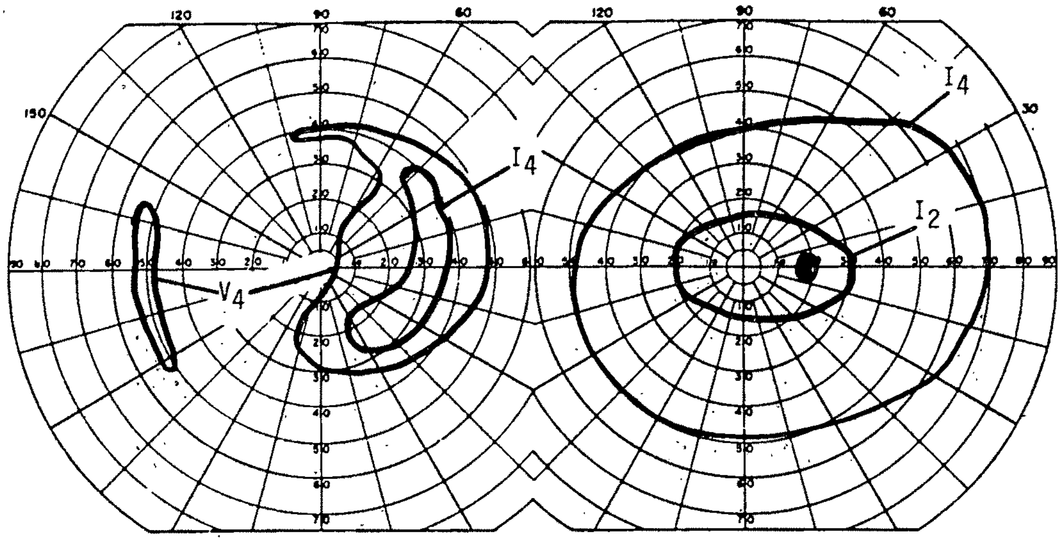

Fig. 1 (DeFrancisco, Savino, and Schatz). Goldmann perimetry reveals a normal right field with a large central scotoma on the left.

periods of increased metabolic stress, such as pregnancy, low carbohydrate intake, infection, alcohol intake, or menstruation; attacks may also be precipitated by various drugs,[1] notably the barbiturates.[6] The course of the disease is highly variable. Patients may die during their first attack or survive numerous attacks over a period of years. The severity of residual defects is dependent upon the magnitude of the attack and the organ systems involved.

Temporary loss of vision as the result of an angiospastic phenomenon during a

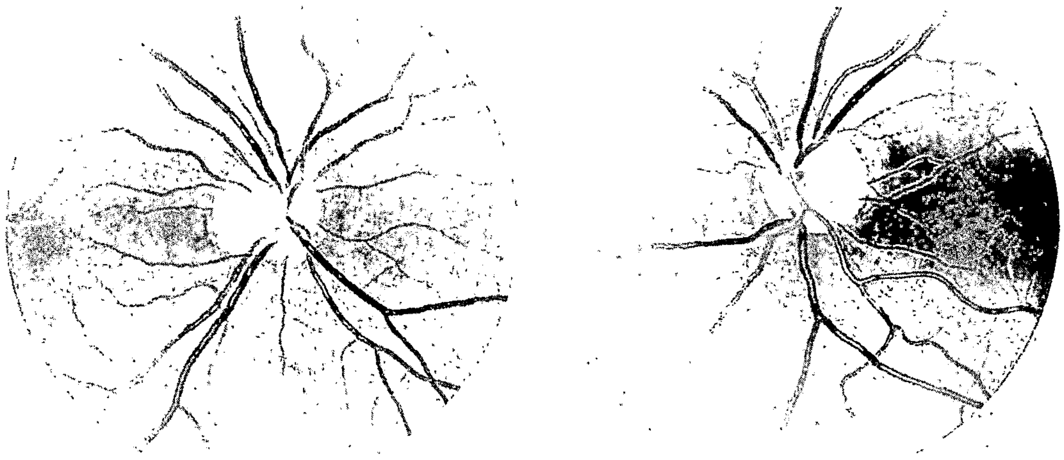

Fig. 2 (DeFrancisco, Savino, and Schatz). The right optic disk is normal. The left disk is pale and the retinal nerve fiber layer is diffusely thinned.

hypertensive phase of an attack of acute intermittent porphyria has been mentioned by several authors.[4,7-9] This angiospastic event may involve retinal blood vessels and has been observed clinically,[4,10] although visual loss without associated retinal artery changes has also been documented.[11] The biochemical defect or toxin that produces vasospasm has not yet been clearly defined.

Wolter, Clark, and Kallet[7] reported ocular abnormalities in a patient with acute intermittent porphyria. Using fluorescence microscopy, they showed fluorescence of the vascular walls especially of the smaller vessels and capillaries. The patient, however, did not have an ophthalmologic examination before death. Additionally, the patient was generally debilitated with malignant hypertension and arteriosclerotic vascular disease. None of the pathologic abnormalities could be attributed specifically to acute intermittent porphyria. Wolter, Clark, and Kallet[7] stated that retinal blood vessel spasm may produce permanent scotomas and blindness. Visual loss also may be caused by extensive bilateral occipital lobe infarction secondary to spasm of both posterior cerebral arteries.[12,13] Martin and Heck[14] reviewed 81 patients with porphyria, 31 of whom had neurologic symptoms or signs. One patient had visual difficulty described only as periodic blindness. Another patient had hemianopia which usually is caused by a posterior circulatory disturbance. Waldenstrom[15] stated that nine patients (4% of cases studied) had visual symptoms, described only as transitory amaurosis without distinction between optic nerve and retinal or occipital lobe amaurosis. Goldberg[16] observed retinal artery spasm in four of 50 patients studied, but apparently partial and temporary loss of visual acuity was noted only in three. No instance of permanent visual loss was recorded and permanent visual loss secondary to retinal or optic nerve ischemia has been rarely documented.

During a severe attack of acute intermittent porphyria, our patient suffered sudden, bilateral loss of vision, retaining only minimal light perception. Her vision gradually returned during the following three weeks, but a central scotoma, which has remained stable and constant, persisted in the left eye. Atrophy of the optic disk that was consistent with the field defect was noted. No well documented reports of optic atrophy and resultant permanent field defects caused by ischemia of the ocular circulation in acute intermittent porphyria exist to our knowledge.

A primary demyelinating lesion which has been reported to affect peripheral nerves and central white matter in acute intermittent porphyria could have permanently damaged a portion of the patient's left optic nerve.[2,4,17] In view of the systemic nature of acute intermittent porphyria, however, specific and limited pathologic involvement of the optic nerves seem unlikely.

We believe that vascular occlusion of the posterior ciliary circulation with accompanying ischemic optic neuropathy was the cause of our patient's visual loss. The normal electroretinogram indicates that the optic nerve, and not the retinal circulation, was the primary site involved.

SUMMARY

A 24-year-old woman developed bilateral blindness after recovery from coma secondary to acute intermittent porphyria. Gradual return of vision in the right eye with a permanent unilateral visual field defect and optic atrophy followed. We believe the pathophysiologic mechanism was spasm of the vessels supplying the optic disk leading to ischemia and infarction of the optic nerve.

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OCULOMOTOR NERVE REGENERATION AFTER ANEURYSM SURGERY

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Intracranial aneurysms may produce oculomotor nerve palsy as the initial clinical symptom[1-4] with or without associated subarachnoid hemorrhage. The most common site of origin for such aneurysms is the internal carotid artery at the origin of the posterior communicating artery, whereas a location at the posterior cerebral artery is less frequent.[1,5] We describe herein a patient who had oculomotor nerve palsy secondary to a posterior cerebral artery aneurysm that did not rupture. Partial recovery occurred after ligation of this aneurysm despite the 12-month duration of oculomotor nerve palsy before surgery. Comparison of light and electron microscopic measurements indicated regeneration of the nerve. The number, density and diameter of axons and their myelin sheaths in the regenerated nerve were compared to the normal oculomotor nerve. Such morphometric evaluations of human peripheral nerves may provide insight into the nature and mechanisms involved in normal myelination,[6,7] as well as in peripheral nerve degeneration and regeneration.[8,9] Others have reported briefly on this study.[10,11]

CASE REPORT

A 57-year-old woman had an acute onset of severe retro-orbital headaches that were accompanied by complete blepharoptosis and diplopia on the right side. Treatment for diabetes was instituted. Glaucoma had been under treatment for several years.

After ten months without improvement, the patient was referred to a neuro-ophthalmologist. Examination revealed absence of headaches, but a complete right oculomotor nerve palsy, both internal and external, and slight exophthalmos. Visual acuity after elevation of the blepharoptic eyelid was normal and visual fields were full in both eyes. Intraocular pressure under treatment with pilocarpine 4% four times a day was normal. A mild hemiparesis was apparent by slight weakness in the left arm hyperreflexia in both the left arm and leg, and a left Babinski response. Computed tomography (Fig. 1) and angiography (Fig. 2) indicated an aneurysm of the right posterior cerebral artery that projected directly into the right cerebral peduncle.

Craniotomy was performed and the aneurysm was found to be firm with a broad origin from the posterior cerebral artery at the junction with the

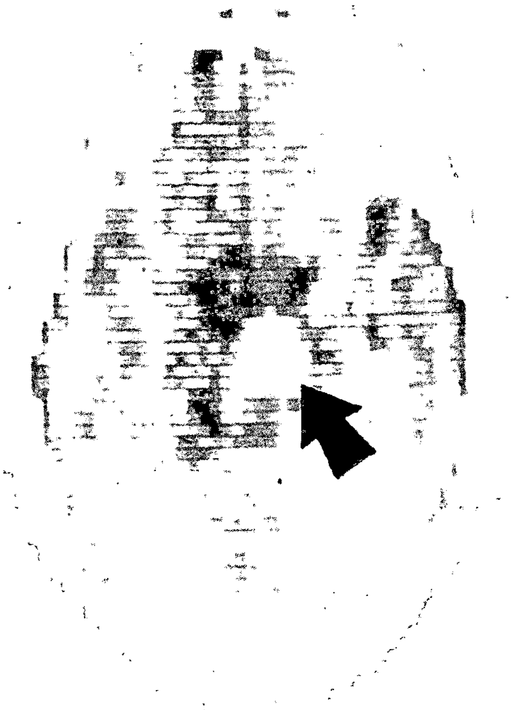

Fig. 1 (Kerns and associates). Computed tomogram revealing a dense mass in the region of the right midbrain (arrow).

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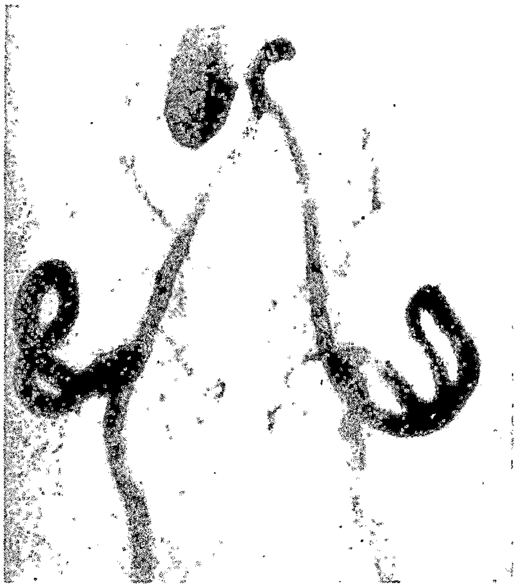

Fig. 2 (Kerns and associates). Angiogram is base view demonstrating the large aneurysm projecting posteriorly from the right posterior cerebral artery into the region of the cerebral peduncle.

posterior communicating artery. It was successfully ligated with a large suture after clipping of the posterior communicating artery. The right oculomotor nerve, passing around the mass of aneurysm, was extremely thin, pinkish, and appeared to be grossly demyelinated. After surgery, the neurologic status was unchanged except for the permanent addition of a complete left homonymous hemianopia. The preoperative hemiparesis gradually improved and the patient was ambulatory.

By two months postoperatively, there was elevation of the right upper eyelid. Oculomotor function slowly returned until one year after craniotomy when there was only slight blepharoptosis of the right upper eyelid in the primary position of gaze. Because the patient had glaucoma and took pilocarpine, this medication was discontinued 72 hours before examination of the pupillary reflexes. The right pupil was slightly larger than the left, but both reacted to light and accommodation. On abduction, the pupillary fissure of the right side narrowed and on adduction the fissure widened. On down gaze to the right, the right upper eyelid retracted and the pupil dilated. On up gaze, the function of the right superior rectus and right inferior oblique muscles was deficient.

At 21 months postoperatively, the patient returned with severe back pain caused by metastatic disease of the lumbar spine. The patient deteriorated rapidly and died two months later of widespread metastatic adenocarcinoma.

Autopsy showed a primary adenocarcinoma of the gall bladder with metatases to liver, lungs, lymph nodes, bones, and adrenal glands. The immediate cause of death was bronchopneumonia. The fixed brain weighed 1,200 g and showed symmetrical cerebral hemisphere with normal gyral configurations and unremarkable leptomeninges.

The saccular aneurysm measured 1.0 × 1.1 × 0.7 cm and projected superoposteriorly into the interpeduncular fossa, where it indented the medial half of the right cerebral peduncle, and was bound to adjacent leptomeninges by fibrous adhesions (Fig. 3). The right oculomotor nerve was closely applied to the aneurysm and was displaced inferiorly by the aneurysm within the interpeduncular fossa; in its extra-axial course beyond the aneurysm, the right oculomotor nerve was distinctly smaller than the left oculomotor nerve, measuring 1.8 mm in greatest diameter as opposed to 2.5 mm for the left oculomotor nerve.

The aneurysm and adjacent posterior cerebral artery were both filled with pale fibrous tissue microscopically consistent with an organized thrombus. A metal surgical clip occluded the right posterior cerebral artery.

The right basis pedunculi showed indentation, compression, and erosion with gliosis in its medial

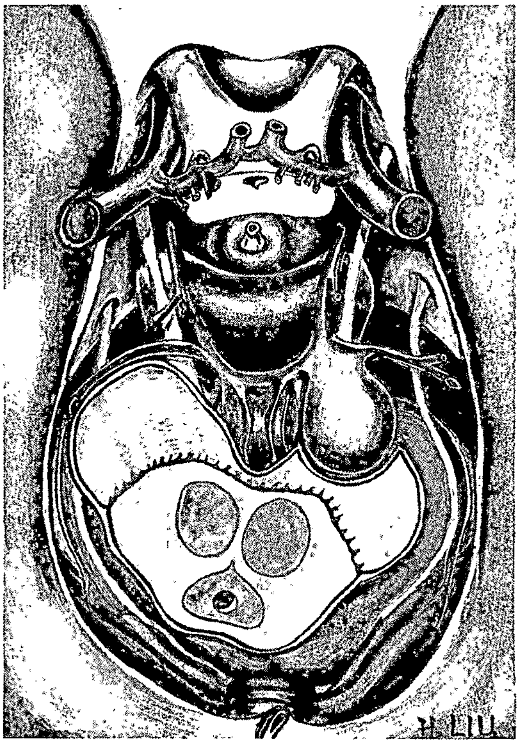

Fig. 3 (Kerns and associates). Artist's depiction of the aneurysm and its anatomic relationships. Anterior is to the top. Note the marked compression of the right cerebral peduncle and the relationship of the right oculomotor nerve.

half, corresponding to the aneurysmal mass. The right corticospinal tracts in pons and medulla were slightly atrophied. The right lateral geniculate body, optic radiations, and portions of the right parahippocampal and medial occipitotemporal gyres, including calcarine cortex, were atrophic and softened with the microscopic appearance of a semicystic healed infarct.

RESULTS

Primary fixation of the brain at autopsy was by immersion in neutral buffered formalin 24 hours after death. The methods of preparation and analysis are otherwise similar to those described by others.[8,9] The right (regenerated) and left (normal) oculomotor nerves were fixed further in a weak aldehyde mixture in cacodylate buffer, osmicated, dehydrated, embedded transversely in Epon-Araldite, and sectioned at 1 μ.

Both nerves were arbitrarily divided into inner core and outer rim portions of equal area (Fig. 4) to test for differential nerve compression effects, and photographs were randomly taken at ×530 magnification from each of these areas. Nerve counts and measurements were made from four randomly selected grids (0.05 mm^2) on each of these four areas (Fig. 5). Fiber counts included all profiles that were identifiable as myelinated axons within the grid area. An estimate of the total number of fibers was derived from these grid counts and the overall area measurements. The sample included about 10% of the total estimated number. Morphometric measurements were made on the first 50 axons having distinct myelin sheaths and nearly spherical profiles from each grid, giving a total of 400 fibers for each nerve. The axon profile (d) was estimated from the perpendicular axes.[12] The myelin sheath thickness was measured at the most uniform point, doubled, added to the diameter (d) to give the overall diameter (D), and the g-ratio (d/D) was then calculated.

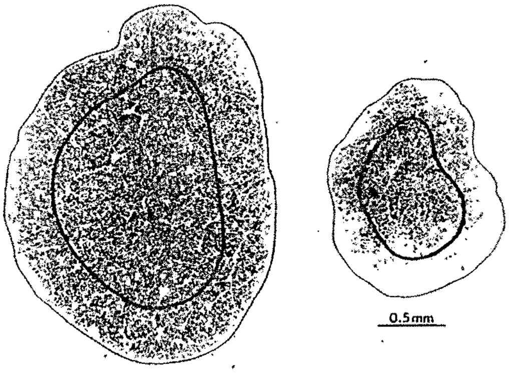

Fig. 4 (Kerns and associates). Comparison of normal oculomotor nerve (left) with the regenerated nerve (right), showing a reduction in the transverse area. The heavy line separates the inner core region from the outer rim in each nerve (toluidine blue stain on 1 μ section, ×37).

The light microscopic appearance of the oculomotor nerve on the normal side was identical in the outer rim and inner core regions (Fig. 4, left and Fig. 5, bottom). The effects of postmortem autolysis were evident, but the morphologic structure of most myelinated axons was sufficient for detailed morphometric comparisons (Table 1). Compared to the normal nerve, the overall area of the regenerated nerve decreased about 62%. The mean density of myelinated axons was slightly increased because of compression in the rim (19%, $P<.10$, Fig. 4, right, and Fig. 5, top left) and even more in the core (57%, $P<.001$, Fig. 5), top right, but the estimated net effect was an overall reduction of about 49% in the total number of fibers (Table 1).

A cluster of small myelinated and unmyelinated fibers was present in the peripheral rim of both the normal and regenerated nerve. The mean diameter of the myelinated axons on the regenerated side was 23% ($P<.001$) less than the normal side (Fig. 5 and Table 2) as determined by two-factor analysis of variance. However, further statistical analysis (Newman-Keuls multiple range test) revealed no significant difference between

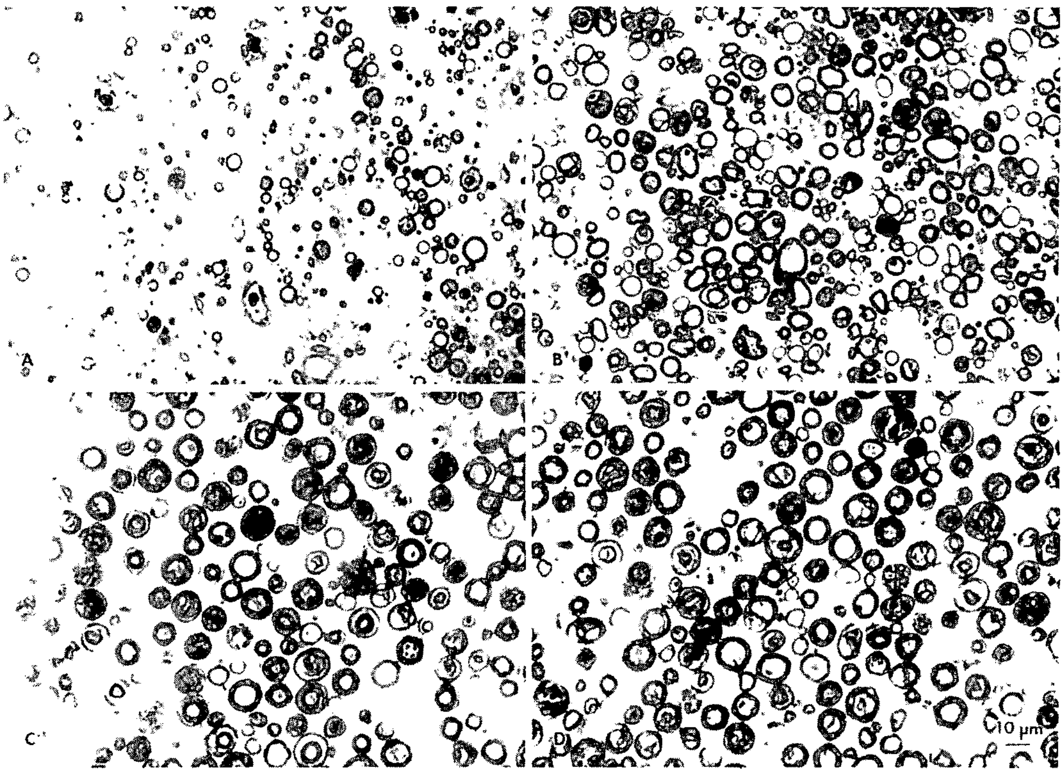

Fig. 5 (Kerns and associates). Light microscopic appearance of the outer rim (top left) and inner core (top right) regions on the regenerated oculomotor nerve compared to the rim (bottom left) and core (bottom right) regions on the normal side. Note the reduction in diameter and myelin sheath thickness on the regenerated side (toluidine blue stain on 1 μ section, ×.350).

the rim and core regions. Similar results were obtained for the g-ratio (d/D), with the control nerve having a mean g-ratio of about 0.64. The overall mean g-ratio of the regenerated nerve (Table 2) was increased significantly by 12% to 0.73 ($P<.001$), but still within the normal range of mean values.[6] A higher mean

TABLE 1
QUANTITATIVE CHANGES IN OCULOMOTOR NERVE

	Normal			Regenerated			
Factor	Rim	Core	Whole Nerve	Rim	Core	Whole Nerve	
Area (mm²)	1.9	1.9	3.8	0.7	0.7	1.4	(−62%)
Density* (No. of fibers/ 0.05 mm²)	303±7	309±4		360±22	486±2		
Estimated No. of fibers	11,500	11,700	23,200	5,000	6,800	11,800	(−49%)

*Mean ± standard error; any two means not spanned by the same underscore are significantly different ($P<.001$), $n=4$.

TABLE 2

QUANTITATIVE CHANGES IN MYELINATED AXONS

Factor	Normal		Regenerated		
	Rim	Core	Rim	Core	
Myelinated axon diameter (μ)*	9.6±0.21	9.1±0.21	7.0±0.14	7.3±0.15	(−23%)
g-Ratio (d/D)*	0.64±.006	0.66±.005	0.73±.005	0.73±.005	(+12%)

*Mean ± standard error; any two means not spanned by the same underscore are significantly different (P<.001), n=200.

g-ratio on the regenerated side indicates that the reformed myelin sheath was proportionately thinner than normal.

There was a negative correlation (r = −0.53, P<.001) beteween the total fiber diameter and the g-ratio in the control nerve (Fig. 6), but the points scattered around a nearly straight regression line (Y = .787 − .014X), with a slightly negative slope (a). A positive correlation (r = 0.53, P < .001) was present in the regenerated nerve. When these latter values were plotted (Fig. 6), the points fit a straight regression line (Y = .600 + .018X) with a slightly positive slope (b). The slopes of the two lines were significantly different (P<.001). The variability of g-ratio values was slightly less for the regenerated nerve (Fig. 6). Both nerves showed a wide range of individual g-ratio values (0.45 to 0.85) so the advantage of greater precision by electron microscopy became less important. Also, variation in the g-ratio along individual fibers (n = 7) measured on adjacent semithin sections was only 2%. These data were evaluated by electron microscopy from a smaller sample with similar results. However, a detailed ultrastructural analysis was not performed because of marginal tissue preservation. Numerous unmyelinated axons and an increased amount of connective tissue were evident in the regenerated nerve (Fig. 7).

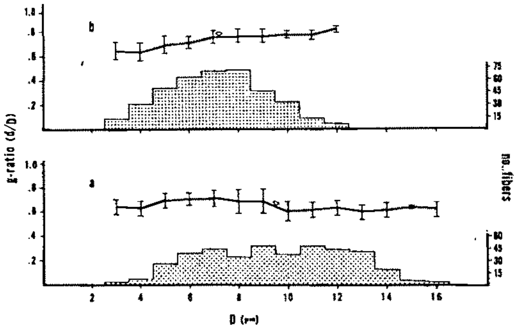

Fig. 6 (Kerns and associates). Plot of the g-ratio (d/D) vs the total diameter (D) for the normal (a) and regenerated (b) oculomotor nerves, with the corresponding means at the arrows on each regression line. The shaded histograms depict the fiber distribution for each nerve. Data for each nerve based on 400 fibers.

DISCUSSION

The prognosis for recovery of oculomotor nerve function is generally excellent if aneurysmal pressure is surgically relieved within the first few days.[3,13,14] In these patients, the lesion is most likely a physiologic conduction block caused by compression rather than axonal disruption. When the duration of paresis lasts weeks or months, regeneration usually requires two to three months.[5,13] Wallerian degeneration may have occurred and actual regrowth of the axons is required. Usually regeneration involves an increase in the number of distal axons because of sprouting, [5,15] instead of a decrease as shown in our case.

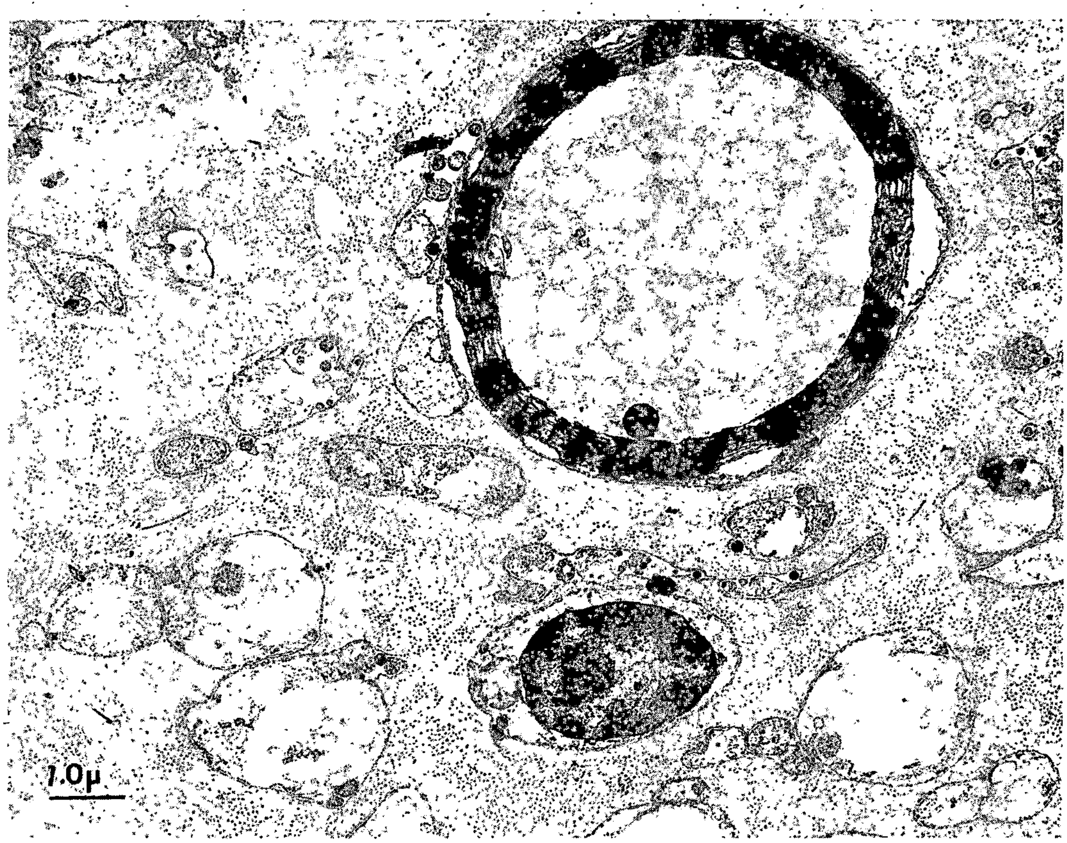

Fig. 7 (Kerns and associates). Ultrastructural appearance of the rim region in the regenerated nerve. A single myelinated axon with a thin myelin sheath is surrounded by many unmyelinated axons and an increased connective tissue endoneurium (electron microscopy, ×10,000).

Delayed regeneration is usually accompanied by a high incidence of aberrant regeneration as described by Walsh[16] and others.[17,18] Others [3,19] have stated that some fiber misdirection always occurs if the nerve lesion is caused by aneurysm or trauma and is of more than six weeks' duration. It is therefore not unusual that our patient showed signs of aberrant regeneration, first apparent two months after surgery and continuing until her death nearly two years later. In the characteristic pseudo-Graefe sign, the eyelid elevates when the affected eye attempts inward or downward gaze, because some axons originally directed to the medial or inferior rectus end up in the elevator muscles. Vertical movements were poorly performed, probably because of simultaneous contraction of the superior and inferior recti by a random blend of the axons directed to these muscles.[16]

There was a significant return of oculomotor nerve function, as indicated by eyelid motility, absence of diplopia, and a normal pupillary response. This was unexpected because of the one-year duration of the preoperative oculomotor nerve palsy, the attenuation of the nerve at surgery, and the marked structural deficits in the regenerated nerve. Lack of correlation between structure and function was noted by Hyland and Barnett[20] in a similar study. The misdirection syndrome has been experimentally produced in monkeys with detailed clinical stages of recovery,[18] but few studies include any histology of the regenerated oculomotor nerve fibers.[20,21]

An estimated number of 23,200 axons in the normal oculomotor nerve in our study agrees with previous estimates of 24,000 by others.[22,23] However, Trautman[24] gave a lower number of axons, a smaller transverse area and a larger mean axonal diameter compared to our values. Most investigators describe the normal human oculomotor nerve as consisting of large extrafusal fibers 10 to 16 μ, with a smaller population of both extrafusal and parasympathetic fibers at 1 to 6 μ.[23–26] This distinct bimodal distribution was not apparent in our material. The peripheral location of a cluster of small presumably parasympathetic fibers[26–28] was confirmed, although they were not included in the random sample for morphometric analysis. Clinically, pressure caused by aneurysm usually affects these smaller pupillary fibers in the periphery.[28,29] The importance of considering regional variations in fiber size and density within a given nerve has been previously noted.[30] In our study, a significant rim or core difference, or both, was found only with fiber density.

In our study, there seemed to be some random sparing of fibers of both large and small diameters, but this is difficult to determine in the late recovery stage.[31] The extent of regeneration is considered more complete one year after crush or compression lesions as compared to nerve section, because the physical alignment of the distal segment is retained.[32] Most investigators believe the recovery is incomplete even after crush lesions, but the greatest reduction in fiber diameter is in the most distal portion of the nerve.[33] Our estimated reduction in fiber diameter by 23% at the site of injury may be conservative.

Numerous studies have shown that myelin sheath thickness is directly related to the axon diameter.[6,7] The relationship is often expressed as a g-ratio of the axon diameter to the overall diameter, and measured values of 0.6 to 0.7 correspond to theoretical predictions.[34] However, the exact nature of the relationship remains unclear. Most assume a curvilinear correlation, with smaller axons having relatively thicker myelin sheaths. Others have reported a horizontal line signifying a constant g-ration, a straight line with either a positive or negative slope, or random scatter.[6,35] Our data of the normal oculomotor nerve suggest a fairly constant g-ratio as the total axon diameter increases.

The slope of the g-ratio in the regenerated nerve shows a significant positive shift and the mean value is higher compared to the normal nerve. In contrast, Schröder[9] showed a negative shift in the g-ratio. Friede[35] found that the g-ratio remained fairly constant at about 0.7 following ligation of the sciatic nerve in young rats. The slopes were the same except at the site of ligation and results were similar after release of the ligation.

The g-ratio variability in the regenerated nerve is less, as compared to the normal nerve, probably because the myelin sheaths have been newly formed. However, Schröder[9] and Friede[35] found just the opposite. Variability in the g-ratio may be related to section thickness, photographic and measurement errors, fixation, species differences, and the peripheral nerve examined.[6,8,9]

There is some indication that the smallest axons have a lower g-ratio, making the plot curvilinear. A greater proportion of these axons in the regenerated oculomotor nerve may correspond to atrophic nerve fibers[9,36] that are either dying or have made inappropriate connections with the eye muscles in aberrant regeneration. Most fibers in the regenerated nerve have a thinner myelin sheath, and this, rather than the decreased axon diameter, causes the reduction in conduction velocity.[34]

Perhaps in our patient the large reduction in area was partially compensated by an increase in fiber density, and the loss of myelinated axons by retention of many unmyelinated fibers.

SUMMARY

A 57-year-old woman had symptoms of oculomotor nerve palsy first appearing one year before successful surgical ligation of a saccular aneurysm arising from the right posterior cerebral artery. During the subsequent postoperative period of two years, oculomotor nerve functions improved as the result of regeneration. Extensive morphometric evaluation of the regenerated nerve was compared to the normal side at the light microscopic level. The affected nerve showed a reduction in the transverse area (62%), estimated number of fibers (49%), and mean diameter of myelinated axons (23%). The normal g-ratio of axon to total diameter was almost constant at 0.64, but on the regenerated side it increased to 0.73. An increase in unmyelinated axons and connective tissue endoneurium was evident at the ultrastructural level. The significance of these marked quantitative changes was compared to the partial return of oculomotor nerve function.

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TERMINAL COURSE OF NERVE SUPPLY TO MÜLLER'S MUSCLE IN THE RHESUS MONKEY AND ITS CLINICAL SIGNIFICANCE

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The superior tarsal (Müller's) muscle is innervated by sympathetic nerve fibers but their course is unknown. They may accompany the motor nerve to the levator palpebrae superioris muscle,[1-2] follow the internal carotid and ophthalmic arteries,[3] or join the ophthalmic division of the trigeminal nerve[3] (Fig. 1). The course is of clinical importance in blepharoptosis surgery.[4] Müller's muscle is present in congenital blepharoptosis[5] and it probably accounts for about 2 mm of eyelid elevation since its paralysis, as in Horner's syndrome, results in this degree of blepharoptosis. The standard blepharoptosis operations remove most or all of Müller's muscle. If the muscle is preserved, it must not be denervated.[6] Many blepharoptosis operations involve cutting the levator muscle. We report herein the results of an investigation designed to learn if this denervates Müller's muscle.

Material and methods

The levator muscle was cut in 16 monkeys and Müller's muscle was examined by electron microscopy for evidence of adrenergic nerve damage, by a modification of the Falck-Hillarp histochemical fluorescence technique which indicates intraneuronal catecholamines in intact adrenergic nerves,[7-8] and pharmacologically by observing changes in eyelid level after various drops.

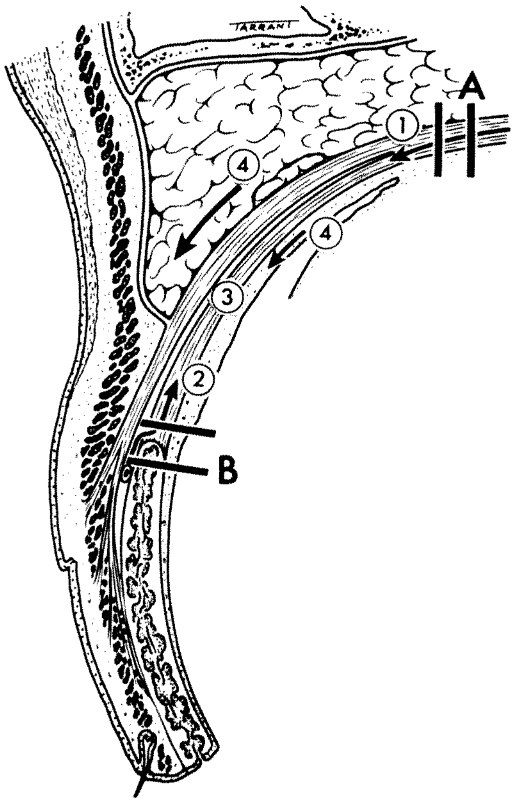

Fig. 1 (Collin, Beard, and Wood). Diagramatic cross-section of upper eyelid showing possible course of the sympathetic nerve supply to Müller's muscle: (1) via the levator muscle; (2) from the perivascular plexus around the marginal artery at the upper border of the tarsus; (3) from the perivascular plexus around other arterioles supplying the muscle; (4) via the sensory nerves. Section of levator muscle above origin of Müller's muscle (A); tissue excised in Fasanella-Servat procedure (B).

Sixteen rhesus monkeys were anesthetized with intramuscular ketamine hydrochloride (10 to 15 mg/lb), which was supplemented by injections of local anesthetic. One upper eyelid was operated on and the other served as a control. A skin

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incision was made in six monkeys through the preseptal skin and orbicularis muscle to expose the orbital septum. This was divided and the pre-aponeurotic fat retracted to expose the muscular body of the levator muscle. The fascial sheath at the medial border of the levator muscle was incised and the muscle picked up with a muscle hook. It was clamped with a hemostat and divided above the origin of Müller's muscle (Fig. 1). The proximal levator muscle was allowed to retract back into the orbit, except in one case, in which a scleral graft was sutured between the cut ends of the muscle. The skin and orbicularis muscle were closed with catgut sutures.

Each monkey had one excision biopsy of Müller's muscle. In different monkeys this was taken at one, two, five, seven, and 16 days after cutting the levator muscle, and three months after the scleral graft. The upper eyelid was everted over a retractor and an incision made through the conjunctiva above the upper border of the tarsus. The conjunctiva was separated from the underlying Müller's muscle with scissors. An incision was made through the tarsus about 1 mm below its upper border into the post-aponeurotic space. This space was opened up, freeing Müller's muscle, which was excised from the muscular levator at its origin. The specimen of Müller's muscle was cut in half vertically. One half was processed for transmission electron microscopy and the other half prepared for the histochemical fluorescence method of demonstrating catecholamines. These specimens were compared with controls from the opposite eyelid taken at the same time by using the same technique and treatment.

The specimens for electron microscopy were fixed with 1% paraformaldehyde-2% glutaraldehyde in 0.2M sodium cacodylate buffer with the addition of 1% sucrose, 0.2% calcium chloride, and potassium chloride. They were then rinsed in 0.2M sodium cacodylate buffer and 0.5M sucrose, postfixed with 2% osmium tetroxide in sodium veronal acetate buffer for four hours, rinsed with several changes of sodium veronal acetate buffer, and en-bloc stained with uranyl acetate stain for three hours. The tissues were rinsed after the staining period, dehydrated in graded alcohol 70 to 100%, treated with propylene oxide, 1:1 propylene oxide and Araldite for two hours, then 2:1 Araldite and propylene oxide overnight, and finally embedded in Araldite. Ultrathin sections for electron microscopy were mounted on grids and stained primarily with 2% uranyl acetate followed by lead citrate. Other sections were stained with potassium permanganate. They were then examined and photographed with an electron microscope.

The specimens for histochemical fluorescence were immersed in liquid Freon 22 for 30 seconds and then placed in liquid nitrogen. They were transferred to a freeze dryer and exposed to a vacuum of 10^{-3} torr at a temperature of −80°C, rising to −60°C for 48 hours. The dried tissue was then exposed to formaldehyde fumes at 80°C for one hour. The formaldehyde fumes were generated from paraformaldehyde, which had been kept at a relative humidity of 70% for several days. The tissue was embedded in purified paraffin and 10-μ sections were cut. Alternate sections were heated to remove the paraffin wax and mounted in liquid paraffin oil. They were examined and photographed through a fluorescence microscope using a BG 12 exciting filter and a Zeiss 50 barrier filter. The remaining sections were stained with hematoxylin and eosin and photographed through a light microscope at similar magnifications.

Two monkeys underwent Fasanella-Servat[9] procedures and a third had a scleral graft placed between Müller's muscle and the upper border of the tarsal plate (Fig. 2). An excision biopsy of

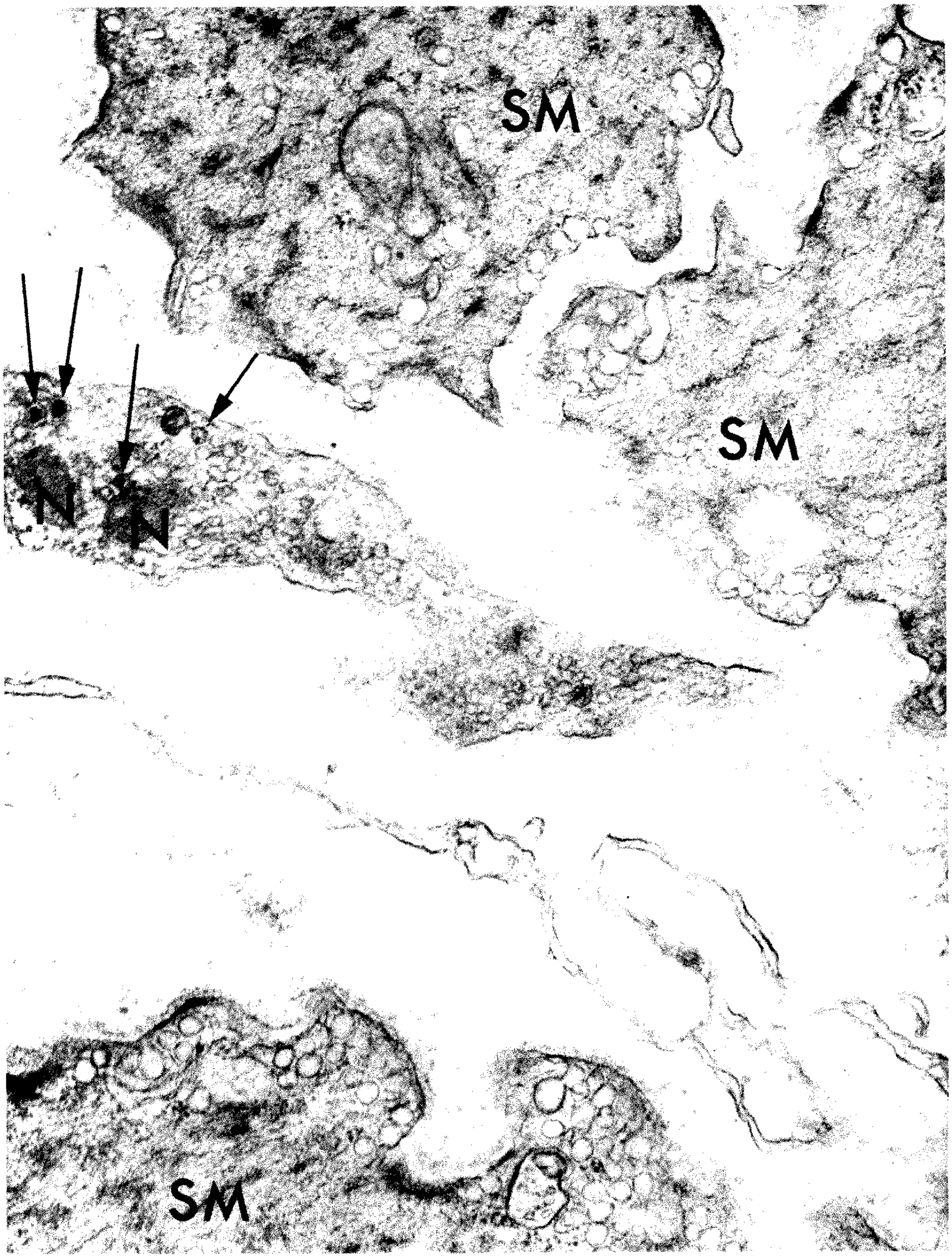

Fig. 2 (Collin, Beard, and Wood). Electron micrograph of normal monkey Müller's muscle cells (SM) surrounding an adrenergic nerve with normal mitochondria (N); arrows point to typical granular vesicles (×28,600).

Müller's muscle was carried out one and seven days after the Fasanella-Servat procedures, and three months after the scleral graft. The specimens and controls were prepared and examined as just described.

Two monkeys had a unilateral superior cervical ganglionectomy. The ketamine anesthesia was supplemented with intravenous sodium pentobarbital, approximately 15 mg/kg of body weight. A vertical skin incision was made under the angle of the jaw and the cervical muscles separated to expose the fascial sheath of the common carotid artery. The cervical sympathetic chain was identified and followed up to the superior cervical ganglion.[10] This was excised close to the base of the skull. Postoperatively the monkeys developed a typical Horner's syndrome. Sections of the excised tissue examined under the light microscope confirmed the presence of sympathetic ganglion cells. An excision biopsy of Müller's muscle was performed on the first monkey after 24 hours. The postganglionic nature of the Horner's syndrome in the second monkey was confirmed five days postoperatively by the lack of pupillary dilatation on that side with 1% hydroxyamphetamine (Paredrine).[11] An excision biopsy of half of Müller's muscle was taken ten days postoperatively and the other half four weeks postoperatively from this monkey.

Another monkey had an identical plication of both levator muscles at the same operation. The plicated levator was cut on one side and not on the other. A full-thickness tuck of muscular levator was created with catgut sutures on each side. The right levator muscle was then cut through the tuck above the sutures. The levator muscle was therefore shortened by a similar amount on the two sides, but divided on the right and kept intact on the left. This produced similar eyelid levels. The effect of 5% guanethidine sulfate eye drops three times a day was then assessed. The levator muscle was cut and sutured in four other monkeys. The changes in their eyelid levels were assessed after the instillation of guanethidine sulfate and various concentrations of adrenaline eye drops, ranging from 0.1% to 2%.

RESULTS

With electron microscopy normal adrenergic nerves were identified in control sections of monkey Müller's muscle by the presence of granular synaptic vesicles containing an electron-dense core, numerous microtubules, neurofilaments, and healthy mitochondria (Fig. 2). The specimen taken 24 hours after the superior cervical ganglionectomy showed evidence of acute nerve damage. Different stages were seen from normal appearance of the nerves to a decrease and loss of the granular vesicles, swelling of the axons with distortion of the mitochondria and surrounding Schwann sheaths, and fewer or absent microtubules and neurofilaments. The cytoplasm of the axons therefore appeared extracted (Fig. 3). The specimens taken ten days and four weeks after the superior cervical ganglionectomy showed more marked damage and there was an almost complete absence of intact nerves. By contrast, intact adrenergic nerves with granular dense-cored vesicles were found in all the specimens taken after the levator had been cut (Fig. 4). These specimens showed some evidence of nerve damage but it was insignificant, as compared with that seen after the ganglionectomies. The specimens taken after the Fasanella-Servat procedures and the scleral grafts also showed both healthy and damaged nerves (Figs. 5 and 6).

Histochemical fluorescence was induced in nerves containing catecholamines. The normal terminal adrenergic nerve fibers in control sections of monkey Müller's muscle produced an intense flu-

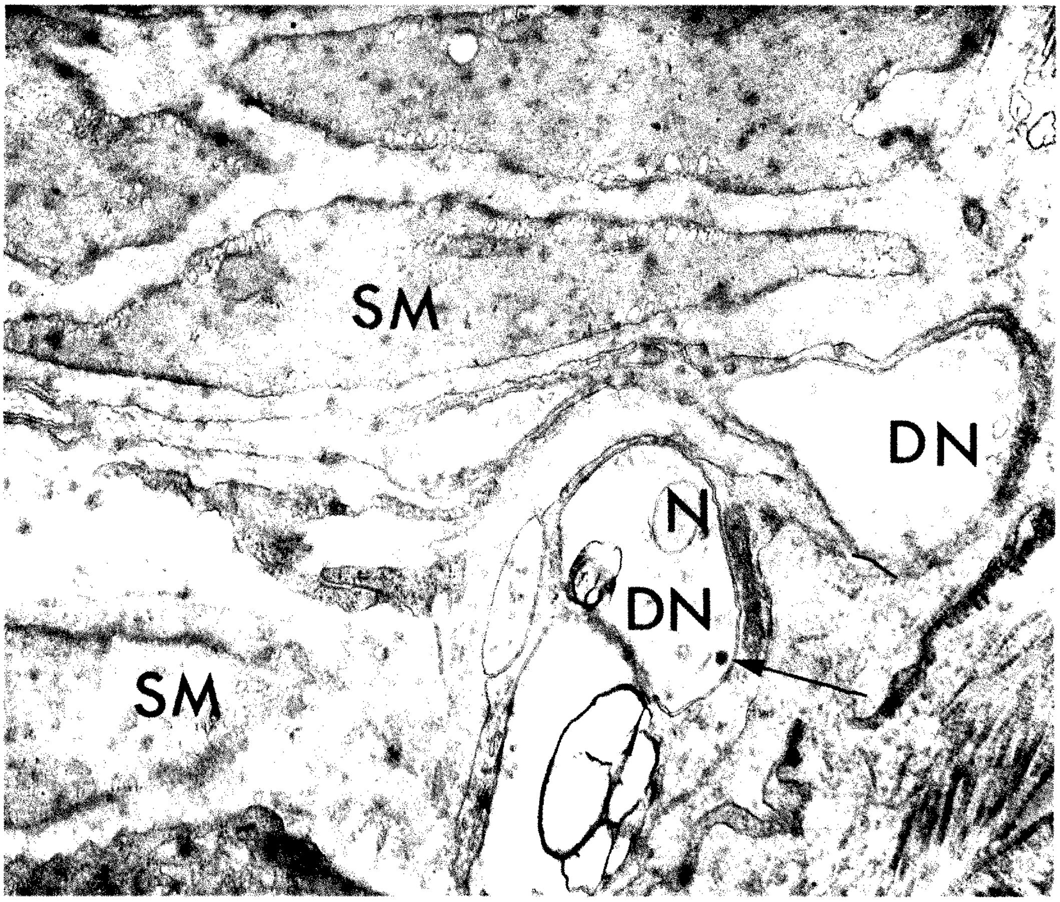

Fig. 3 (Collin, Beard, and Wood). Electron micrograph of monkey Müller's muscle cells (SM) surrounding damaged adrenergic nerves (DN) with arrow pointing to granular vesicle. Distorted mitochondrion (N). Twenty-four hours after superior cervical ganglionectomy (× 13,250).

orescence (Fig. 7, left). Alternate sections from the same block stained with hematoxylin and eosin showed the corresponding light microscopic appearance (Fig. 7, right). The sections were adjacent but the appearances were not identical because the sections were treated in different ways and photographed through different microscopes. Specific fluorescence was present in all the sections of Müller's muscle taken at various periods after the levator muscle was cut (Fig. 8) and after the Fasanella-Servat procedures (Fig. 9). It did not appear as intense as in the control sections but it was specific. This was confirmed by reducing random sections with 0.03% sodium borohydride in 95% isopropyl alcohol. The fluorescence was abolished but was regenerated by reexposure to formaldehyde gas.[12] Specific fluorescence therefore persisted in adregenergic nerve terminals in close relation to Müller's muscle, both after the levator muscle was cut and after the Fasanella-Servat procedures. This confirmed the electron microscopy finding that neither procedure totally denervated Müller's muscle. However, it was not possible to

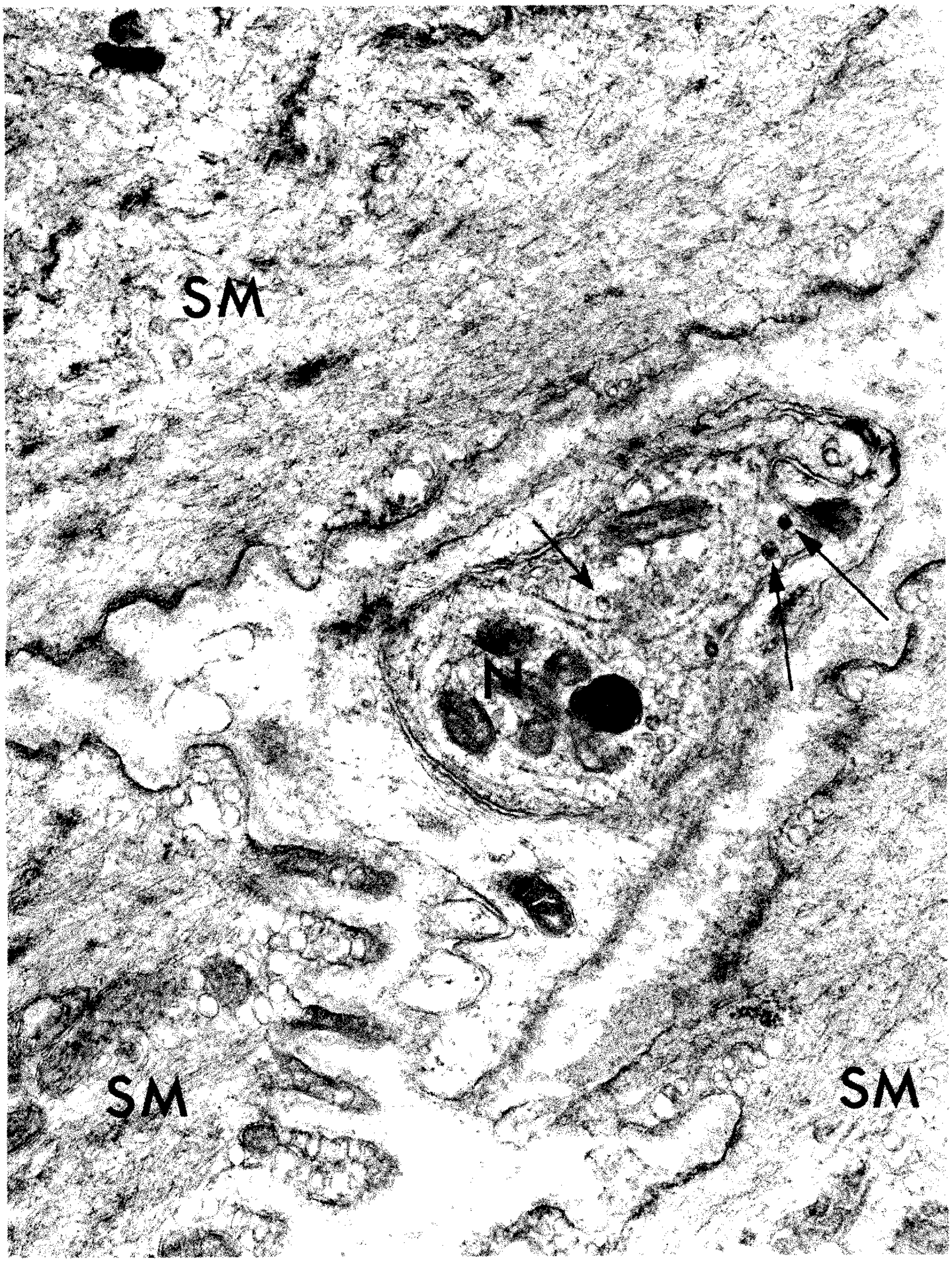

Fig. 4 (Collin, Beard, and Wood). Electron micrograph of monkey Müller's muscle cells (SM) surrounding an intact adrenergic nerve with arrows pointing to typical granular vesicles. Normal mitochondria (N). One week after cutting levator muscle (×24,800).

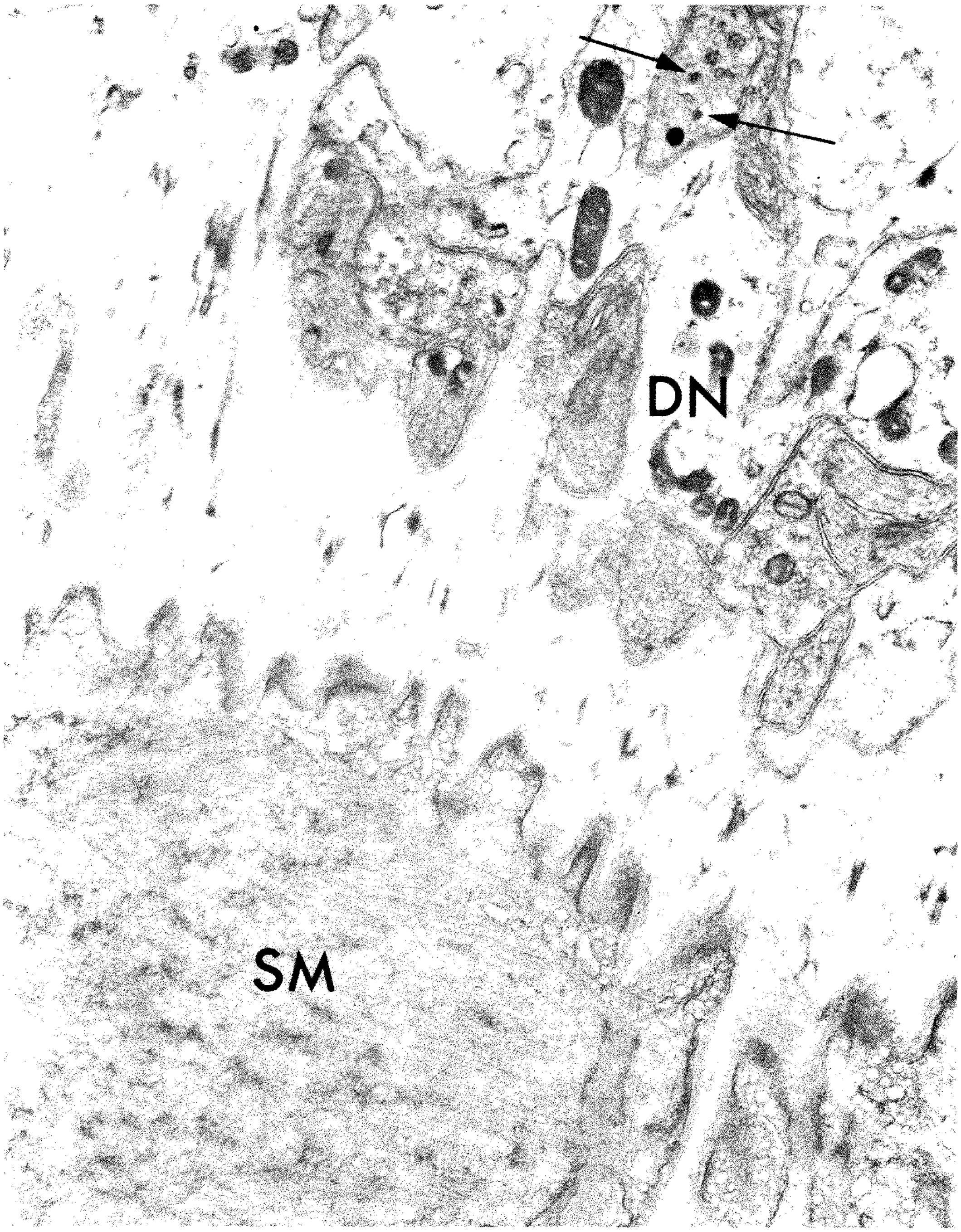

Fig. 5 (Collin, Beard, and Wood). Electron micrograph of monkey Müller's muscle cells (SM) surrounding both damaged nerves (DN) and intact adrenergic nerves with arrows pointing to granular vesicles, twenty-four hours after Fasanella-Servat procedure (×16,850).

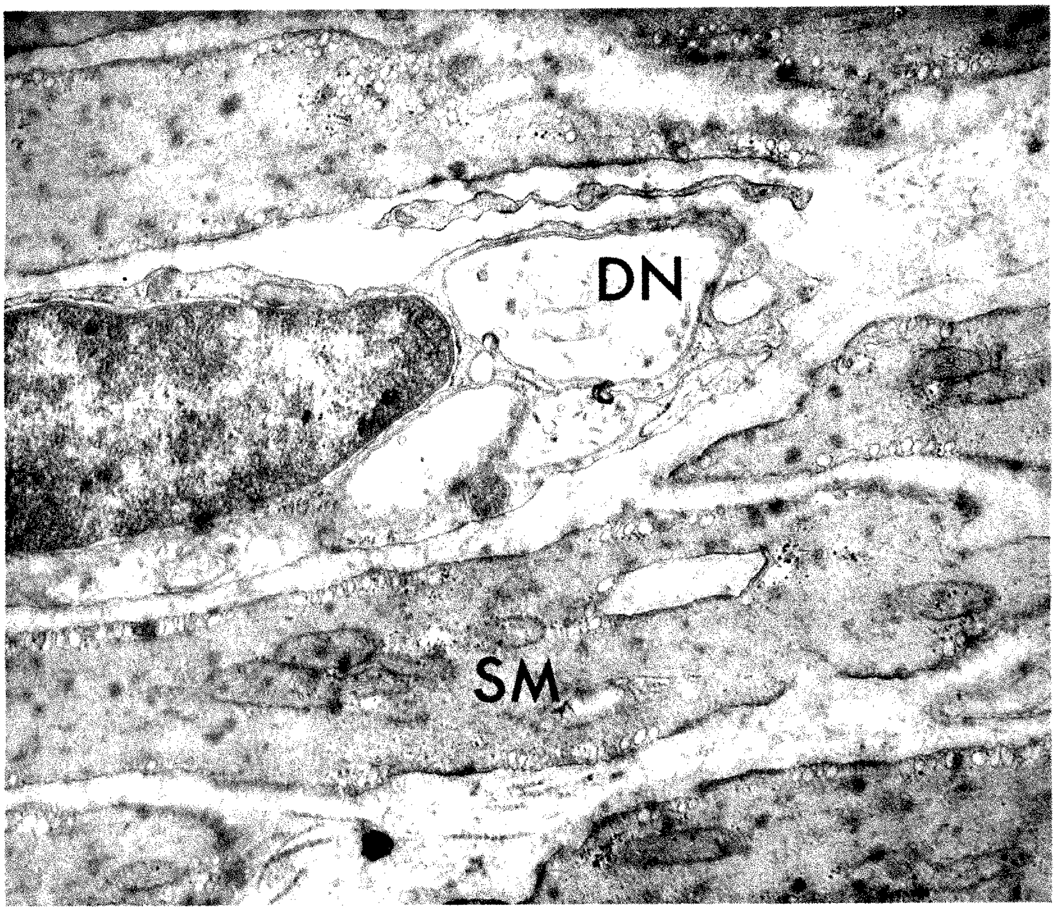

Fig. 6 (Collin, Beard, and Wood). Electron micrograph of monkey Müller's muscle cells (SM) surrounding damaged nerves (DN). One week after Fasanella-Servat procedure (×13,250).

relate any quantitative change in fluorescence to any degree of partial denervation, which probably did occur.

Pharmacologically induced changes in eyelid level were difficult to assess and more difficult to document photographically in monkeys. Any change was most easily detected when the eyelids were at approximately the same level before any drops were instilled. There was no apparent hypersensitivity of Müller's muscle to weak solutions of adrenalin after the levator muscle was cut or after a Fasanella-Servat procedure.

Guanethidine induced a partial blepharoptosis in the monkey with a cut and plicated levator muscle (Fig. 10). It has been used in humans as a sympathetic blocking agent to correct eyelid retraction.[13] Its locus and mechanism of action depends on an intact adrenergic nerve supply.[14] This suggests that cutting the levator muscle did not sufficiently interrupt the adrenergic nerve supply to Müller's muscle to prevent the action of guanethidine. A similar response was obtained in the four monkeys with a cut and sutured levator muscle. Guanethidine also induced a partial blepharoptosis after the Fasanella-Servat procedures. It af-

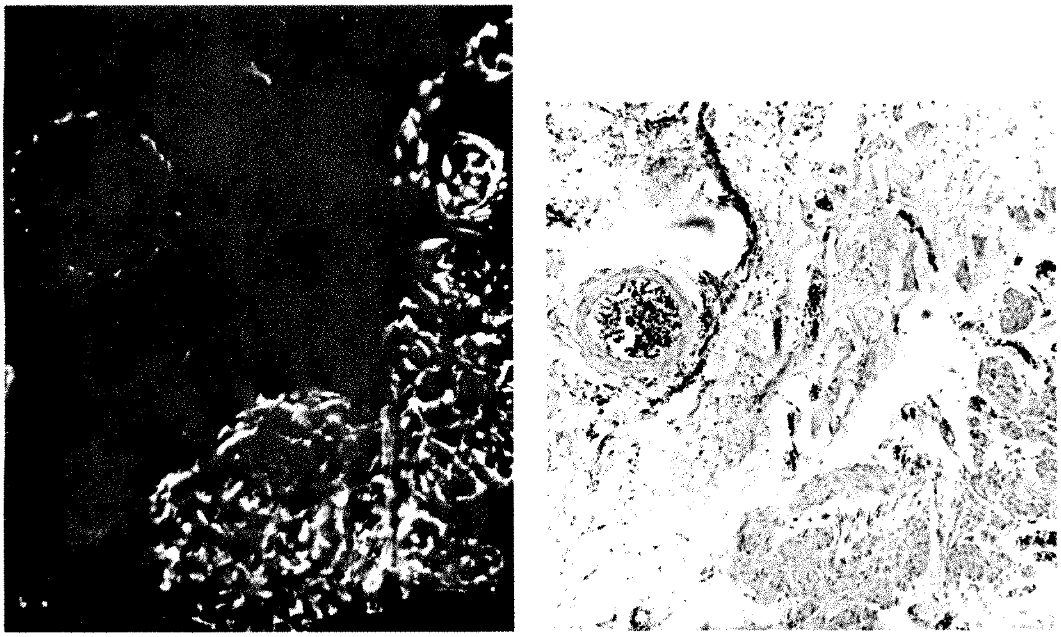

Fig. 7 (Collin, Beard, and Wood). Left, Fluorescent micrograph of normal monkey Müller's muscle treated by the Falck-Hillarp technique. Fluorescence is seen in the vessel wall as well as around the Müller's muscle cells (×80). Right, Adjacent section showing the corresponding light microscope appearance (hematoxylin and eosin, ×60).

fects the sympathetic nerve supply to all orbital smooth muscle and could theoretically cause an apparent blepharoptosis by inducing enophthalmos, but no enophthalmos could be measured. The evidence therefore supported the electron microscopy and histochemical fluorescence findings that Müller's muscle was not totally denervated by either cutting the levator muscle or by a Fasanella-Servat procedure.

DISCUSSION

Adrenergic nerves can be identified electron microscopically by the presence of granular synaptic vesicles containing

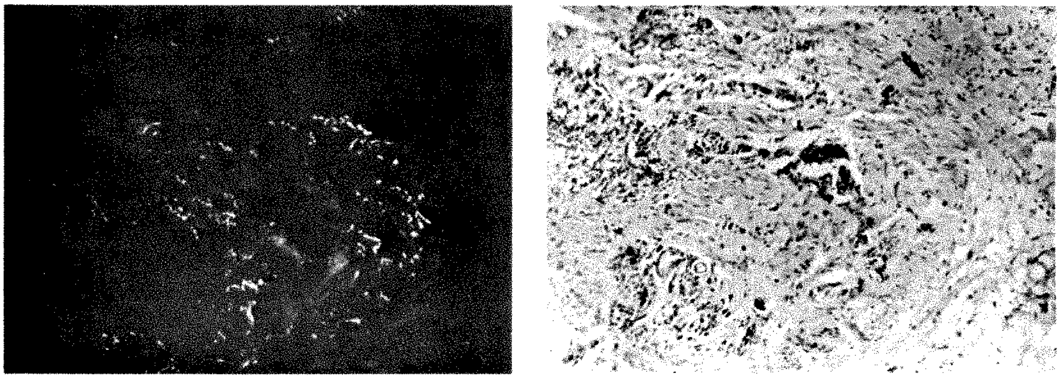

Fig. 8 (Collin, Beard, and Wood). Left, Fluorescent micrograph of monkey Müller's muscle one week after cutting the levator muscle (×80). Right, Adjacent section showing light microscopic appearance (hematoxylin and eosin, ×80).

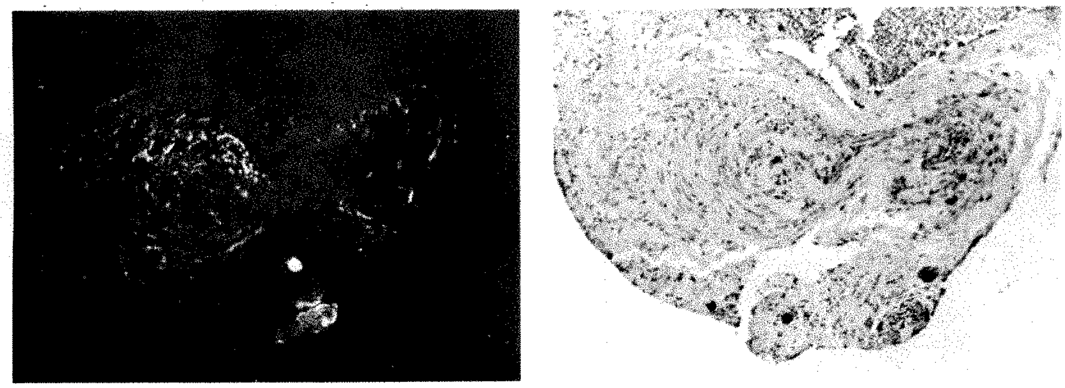

Fig. 9 (Collin, Beard, and Wood). Left, Fluorescent micrograph of monkey Müller's muscle one week after a Fasanella-Servat procedure (×80). Right, Adjacent section showing light microscope appearance (hematoxylin and eosin, ×80).

an electron-dense core. Healthy looking nerves containing these vesicles were present between Müller's muscle cells, both after cutting the levator muscle and after the Fasanella-Servat procedures. This is strong evidence that Müller's muscle was not totally denervated by either procedure. When a nerve is cut it shows degenerative changes. Nerves showing such degenerative changes can still be identified as adrenergic before all the granular synaptic vesicles are lost. Acute nerve damage is more distinctive than chronic nerve damage.[15,16] For this reason early biopsy specimens were taken. There was evidence of nerve damage both after cutting the levator muscle and after the Fasanella-Servat operations, suggesting that both procedures had damaged some of the adrenergic nerve supply to Müller's muscle. The appearance of nerve damage can be simulated by poor fixation techniques, and therefore conclusions based on electron microscopic evidence of nerve damage should be treated cautiously.

Possibly not enough time had elapsed for a loss of induced fluorescence in our early excision biopsy specimens. Variations in the rate of this loss after axotomy are considerable.[17,18] However, it should have been lost by four days after division of the sympathetic nerve supply. The presence of induced fluorescence in

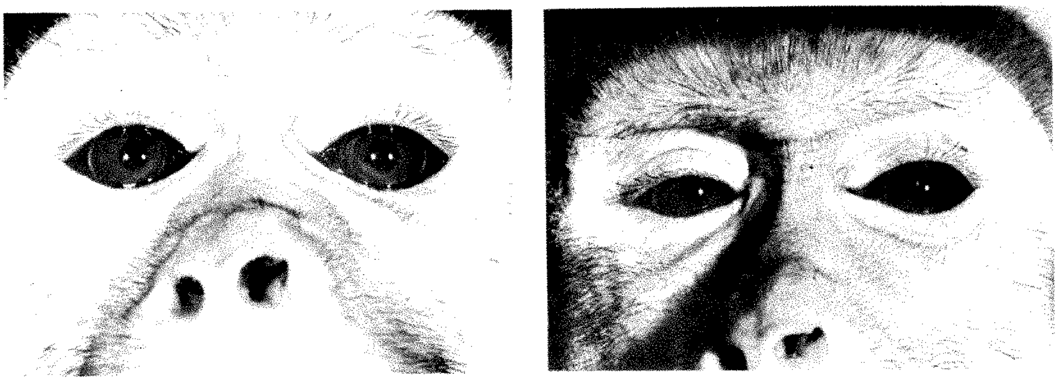

Fig. 10 (Collin, Beard, and Wood). Left, Monkey after plicating both levator muscles and dividing the right levator muscle through the tuck. Right, Same monkey after guanethidine eye drops had induced a right blepharoptosis.

our later specimens is strong evidence that cutting the levator muscle did not totally denervate Müller's muscle. Fluorescence was not induced in the cat's nictitating membrane four weeks after superior cervical ganglionectomy.[19] Fluorescence should have been absent from our specimens taken ten days and four weeks after superior cervical ganglionectomy. The sodium borohydride reduction and regeneration test[12] suggested that our technique did induce specific fluorescence. The persistence of some fluorescence probably occurred because the ganglionectomy was incomplete and part of the upper pole of the ganglion was not excised. The morphological evidence suggests that this is where the cell bodies for the sympathetic supply to the orbital smooth muscle are situated.[20] An alternative possibility for the persistence of intact adrenergic fibers in a monkey is that there may be scattered ganglion cells along the course of the internal carotid and cerebral arteries, which would not be removed by a superior cervical ganglionectomy.[21]

Jones[22] showed that Müller's muscle in man has developed phylogenetically from the capsulopalpebral head of the primitive rectus muscle. In man and the rhesus monkey Müller's muscle has remained as the smooth muscle continuation of the levator muscle. Its nerve supply could travel through the levator muscle in the capsulopalpebral head of the primitive superior rectus muscle. Our experiments show that cutting the levator muscle does not totally denervate Müller's muscle.

The smooth muscle of the nictitating membrane in the cat has developed from the capsulopalpebral head of the primitive medial rectus muscle. Acheson[23] showed that it arises from the fascial sheath of the medial rectus muscle and is continuous with the superior tarsal muscle. Thompson[24] showed that the sympathetic nerve supply to this smooth muscle can be traced with the ophthalmic division of the trigeminal nerve and then with the nasociliary and infratrochlear nerves. He found no evidence that the oculomotor nerve carried any postganglionic sympathetic nerve fibers to the muscle. Morphologically therefore, in man the sympathetic supply to Müller's muscle may be carried with the sensory nerves. We have not disproved this by our experiments. Sensory nerves must have been divided when the levator muscle was cut and we did find evidence of sympathetic nerve damage in these sections. However, we also found evidence of damaged sympathetic nerves between Müller's muscle cells after the Fasanella-Servat procedures. Although sensory nerves would again be sectioned by such procedures, any afferent sympathetic nerves to Müller's muscle that were carried by these sensory nerves would probably already have supplied the muscle. It therefore seems unlikely that all the sympathetic nerve supply to Müller's muscle is carried by the sensory nerves.

Embryologic studies suggest that Müller's muscle and the aponeurosis differentiate in situ from the mesoderm that forms the upper eyelid, and that the levator muscle grows forward from the back of the orbit to join the aponeurosis and Müller's muscle.[25] This is supported by the consistent presence of normal Müller's muscle in all cases of congenital blepharoptosis even if the striated levator muscle itself is defective.[5] The vascular supply of the upper eyelid also develops from local mesoderm and the arterioles are surrounded by a sympathetic plexus of nerves. A Fasanella-Servat procedure would cut some of the arteriolar supply to Müller's muscle from the vascular arcade at the upper border of the tarsus. This did produce damage to sympathetic nerves between Müller's muscle cells, which suggests that the sympathetic nerve supply to Müller's muscle travels with the arterioles that supply the muscle.

The course of the sympathetic nerve supply to Müller's muscle may not be important clinically. If the nerve supply was cut, re-innervation might soon take place as the result of the regenerative capability of the sympathetic system. Butson[26] showed considerable regeneration of the cervical sympathetics in rabbits and cats after crushing or dividing the preganglionic fibers. No regeneration occurred in rabbits after sectioning the postganglionic fibers, although the fibers did seem to be regenerating. Olson[27] in examining the regenerating sympathetic axons in the rat sciatic nerve found that a compression injury to these postganglionic fibers did not prevent regeneration, but cutting the nerve did because of scar tissue formation. The regenerating fibers were not able to cross this scar tissue barrier during the period of observation. If a surgical procedure did cut all the sympathetic nerve supply to Müller's muscle, any significant re-innervation of the muscle is unlikely.

We have shown that neither cutting the levator muscle nor a Fasanella-Servat procedure totally denervates Müller's muscle. The surviving nerves are capable of collateral sprouting as suggested by Edds.[28] This does occur in the sympathetic system and has been demonstrated after partial denervation of the nictitating membrane in the cat.[29] Collateral sprouting should make up for any reduction in the nerve supply to Müller's muscle that does occur with either procedure.

The postganglionic sympathetic nerves arrive at Müller's muscle from several peripheral pathways. They may reach the muscle through the levator muscle, via the sensory nerves, or with the arterioles (Fig. 1). We believe that the arteriolar route is the most likely. Our experiments show that in the rhesus monkey, Müller's muscle is not totally denervated either by cutting the levator muscle or by a Fasanella-Servat procedure. The anatomy of man and the rhesus monkey may not be identical; however, if these results are applicable to man, it should be possible to cut the levator muscle and preserve both Müller's muscle and its nerve supply during blepharoptosis surgery.

SUMMARY

Electron microscopic, histochemical fluorescence, and pharmacologic evidence suggested that, in the rhesus monkey, Müller's muscle was not totally denervated either by cutting the levator palpebrae superioris muscle or by a Fasanella-Servat procedure. We examined the terminal course of the sympathetic nerve supply and the potential for perserving Müller's muscle in blepharoptosis surgery.

ACKNOWLEDGMENT

Mary Grillo, Ph.D., Mrs. Diane Ralston, and Mr. Ben Goldfeller provided the equipment and animals for this study.

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NOTES, CASES AND INSTRUMENTS

REMOVAL OF PRERETINAL BLOOD DURING VITRECTOMY

JAMES D. KINGHAM, M.D.
Tucson, Arizona

After the removal of vitreous, a dense lamina of tenacious preretinal blood occasionally remains over the macula and posterior pole. I remove this blood by a modification of Machemer's[1] original method of the removal of vitreous blood pools. First, the cutting tip of the Ocutome[2] is removed from the eye; I then insert a 38-mm 21-gauge needle attached to an infusion bottle into the eye through the same sclerotomy site used for the cutting tip. The infusion used during the cutting procedure is disconnected and becomes a port of exit for the mixture of blood and balanced salt solution. A short period of irrigation with balanced salt will remove the preretinal blood. To prevent hole formation by the irrigating stream, the needle must not be placed too close to the retina and the pressure must not be too high. I have used this technique in seven patients to remove preretinal blood without harmful effects.

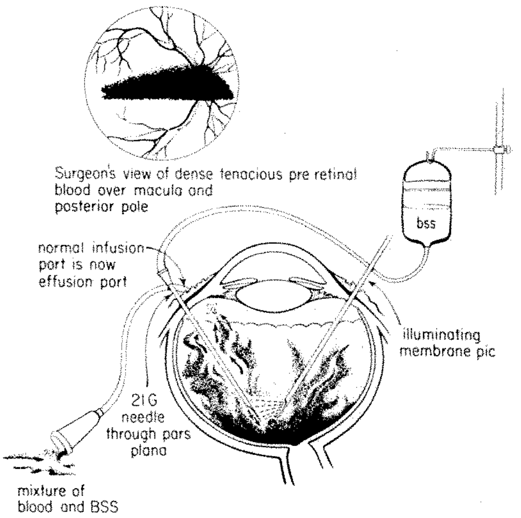
Surgeon's view of dense tenacious pre retinal blood over macula and posterior pole
bss
normal infusion port is now effusion port
illuminating membrane pic
21G needle through pars plana
mixture of blood and BSS

Figure (Kingham). Top, Surgeon's view through operating microscope. Bottom, Infusion is directed on lamina of preretinal blood. Mixture of blood and balanced salt solution egress via needle infusion set.

SUMMARY

To remove preretinal blood from the posterior pole, balanced salt solution is irrigated into the eye through a 21-gauge needle. The needle previously used for infusion is used as a port of exit. I have treated seven patients with this method without harmful effects.

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AMERICAN JOURNAL OF OPHTHALMOLOGY

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Published monthly by the Ophthalmic Publishing Company
Tribune Tower, 435 North Michigan Avenue, Chicago, Illinois 60611

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HAPPY ANNIVERSARY TO THE ARCHIVES

This year marks the centennial anniversary of the Archives of Ophthalmology, for it was in 1879 that the Archives of Ophthalmology severed its connection with Otology and continued as a separate publication. Albert Einstein and J. C. Maxwell, the English physicist, were born in 1879. The first telephone exchange in London was established and the first electric tram was exhibited. Herbert Spencer published "The Principle of Ethics," and Henry James published "Daisy Miller." The French Panama Canal Company was organized and Alsace Lorraine was declared an integral part of Germany.

The Archives of Ophthalmology and Otology was established in 1869 by Herman Knapp of New York City and S. Moose of Heidleberg. Herman Knapp had arrived in America but a year earlier at the age of 36. The American edition was published by William Wood and the German edition was published simultaneously by C. S. Müller. Articles submitted in German were published in English in the United States edition, and those submitted in English were published in German in the German edition. In 1879 the two journals bifurcated into the Archives of Ophthalmology and the Archives of Otology.

The first quarterly issue of the Archives of Ophthalmology appeared in May 1879. The preface stated:

> This division has been made both for the convenience of many readers who practice either ophthalmic or aural surgery exclusively and for the sake of doing justice to the growing importance of the literature in both departments. The English and German editions contain the same original papers either in the language of the authors or in faithful translation. This plan will be essentially continued, but if the contributions become too numerous some papers will be abridged in translation.
>
> Profiting by the past experience and relying on prominent men as regular contributors the Editors feel assured of being able to furnish to their readers a good journal presenting the actual state and practice of the ophthalmic sciences and art in all of their branches. In their selection the Editors will give preferences to those original papers in which speculation and theory are

subordinate to direct observation and fact. Their greatest gratification will be to hear the readers continue to say, "What we read in your journal we can make use of."

The simultaneous American and German edition appeared regularly until 1921 when the Archives of Ophthalmology proceeded as an independent, English edition. Herman Knapp died April 30, 1911, after contributing more than 150 articles to the Archives. In Herman Knapp's obituary Gruening stated, "As an Editor he would on his own responsibility correct erroneous and omit unscientific statements." This course sometimes gave rise to acrimonious correspondence, which however, did not alter his intent to maintain the scientific standards of his publication. His son, Arnold Knapp, became Chief Editor of the Archives of Ophthalmology in January 1911. He was then 42 years old and had begun publishing in the Archives in 1899 with his paper, "A case of extensive chronic emphyema of the frontal and ethmoidal sinuses with excophthalmus operation recovery."

The late 1920s proved to be a difficult time for all medical publications. On Jan. 1, 1929, the American Medical Association became the publisher. The Editorial Board then consisted of Arnold Knapp as Chief Editor; William Zentmeyer, George S. Derby, Sanford R. Gifford, Francis Heed Adler, and John Herbert Waite. On Dec. 31, 1948, Knapp resigned as Chief Editor. He was succeeded by Francis Heed Adler whose initial Editorial Board consisted of William Zentmeyer, William L. Benedict, Frederick C. Cordes, and David Cogan. Subsequently the Editorial Board increased in size to include Kenneth C. Swann, William F. Hughes, Jr., Jonas S. Friedenwald, and John H. Dunnington. Adler also appointed a Board of Associates, each member to serve for three years and to be responsible for preparation of the annual review of ophthalmology. In 1960 the American Medical Association established a policy of a 10-year maximum term for any member of the Editorial Board of any AMA publication. Dr. Adler was replaced as Editor by David Cogan. However, in a remarkable trade, Dr. Adler's name appeared in the June 1960 number of the AMERICAN JOURNAL OF OPHTHALMOLOGY as a consulting editor, a position retained until Derrick Vail was no longer editor-in-chief. After David G. Cogan, the Archives was edited by Henry Allen and currently is edited by Frederick C. Blodi.

We thus salute the beginning of the second century of continuous publication by the Archives of Ophthalmology and compliment the past and present editors who have provided American and international ophthalmology with an outstanding record of opthalmic progress.

FRANK W. NEWELL

OBITUARY

Frank B. Walsh, M.D.

1895-1978

It is with great sadness that I begin this obituary for Frank B. Walsh, for he was one of the truly fine people of this world.

Frank was born in Oxbow, Saskatchewan, Canada, Oct. 18, 1895. During his boyhood he developed an interest in outdoor life, hunting in the woods of Saskatchewan, an activity he continued into his later years. As a young man he was a member of the Oxbow hockey team. He was an avid, capable, left-handed golfer. At the age of 80 he was still able to play 18 holes with a score of 82. In 1915 he joined the Canadian Army together with eight other members of his hockey team. At the end of the war, there were only five. After the war Frank returned to Canada and attended the University of Manitoba, where he received his M.D. degree in 1921. He then went into general practice in Estevan, Saskatchewan, and became a

Frank B. Walsh, M.D.

Fellow of the Royal College of Surgeons of Edinburgh in 1928. Two years later he abandoned general practice and entered the residency at the Wilmer Institute in 1930 at the age of 35.

Often, a relatively minor incident tells more about the character of an individual than a long recitation of his accomplishments. There was a story frequently told about Dr. Walsh's independence, which is not meant to reflect unfavorably on either of the individuals in this story. It occurred during the chairmanship of Dr. Wilmer, who had been a general in the Army Air Force during World War I and in charge of aviation medicine. During this tour of duty he acquired some military attitudes and disciplines. One of these was to have the chief resident meet him at the front door of the Wilmer Institute and escort him down the corridor to his office. When Dr. Walsh became resident he thought this unnecessary and refused to meet the Chief. In those days the Head of the Department was treated with extreme respect and no one dared question his word. This was particularly true of Dr. Wilmer. Therefore, it took great courage on Dr. Walsh's part to show this independence. Dr. Wilmer then responded by refusing to make rounds with Dr. Walsh and avoiding him in other ways. Again, it is to Dr. Walsh's great credit that he saw that his display of independence was disrupting things and because of this he had the humility to return to meeting Dr. Wilmer in the morning at the front door.

Dr. Walsh was always extremely fond of teaching, and was certainly a superb instructor. During his residency he began conferences each Saturday morning on neuro-ophthalmology, and until his terminal illness seldom missed a meeting. It was also at his suggestion that the Wilmer Residents meetings, which are held each year in the spring, were begun. Because of his great love for teaching and instruction of the younger staff, he was affectionately known as "Pappy" by the house staff and students.

After the completion of his residency he joined the fulltime staff of the Wilmer Institute, and in 1937 he began his famous book of medical ophthalmology, "Clinical Neuro-ophthalmology." This classic in the field of our specialty was first published in 1947.

In 1945, personal necessity caused him to leave the fulltime faculty of the Wilmer Institute and enter private practice. A year or two later he joined Charles Iliff in a partnership that lasted until 1957. He then returned to Wilmer to become a geographic fulltime member of the faculty, a position he maintained until the time of his death.

The second edition of "Clinical Neuro-ophthalmology" was published in 1957, and a third and enlarged edition was published with the collaborative editorship of one of his former students, William F. Hoyt.

Until the middle to late 1950s it was the custom at Johns Hopkins for only the chairman of a department to receive the rank of full professor. This precedent was broken in about 1957, and Dr. Walsh was one of the first non-chairmen to be elevated to the rank of full professor in 1958. In 1962 he became emeritus. However, his emeritus status was in name only, for he continued his teaching activities with the same zeal and vigor he had in previous years. He also continued his research in the field of neuro-ophthalmology and in 1973 he published "Neuropathology of Vision—An Atlas," with his collaborators, Richard Lindenberg and Joel Sacks. At the time of his death he was still working on two other books, "Abnormal Ocular Movements" and "Neuropathology of Vision" in collaboration with Richard Lindenberg.

He was unquestionably one of the finest teachers of his generation. There is hardly a neuro-ophthalmologist in this country who was not trained either by Dr. Walsh or by one of his disciples. He is rightfully called, "The father of neuro-ophthalmology."

Because of his many contributions to our specialty, he was awarded the American Medical Association Prize in Ophthalmology, the Howe Medal of the American Ophthalmological Society, the Howe Medal of the Buffalo Ophthalmological Society, the Proctor Medal and Award, the Carl Liebrecth Prize in Hamburg, Germany, and was the first recipient of the Jules Stein Award, presented by Research to Prevent Blindness, Inc., to the person who had contributed more to the advancement of ophthalmology than anyone else in his time. In 1972, Johns Hopkins awarded him an Honorary Doctor of Law degree.

If one were forced to pick out this fine gentleman's greatest virtue or accomplishment it would be difficult to do so. Unquestionably he was one of the great teachers in medicine of our time. His scientific papers have been excellent; his books are known throughout the world as the authoritative work in neuro-ophthalmology. They differ from the usual textbook in that there is hardly a page on which there is not an original contribution that resulted from the clinical observations of Dr. Walsh. Great as all these academic contributions have been, Dr. Walsh's simplicity, honesty, and love of his fellow man and his insistence on justice, made him a truly great person.

A. EDWARD MAUMENEE

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on $8^1/_2 \times 11$-inch bond paper, with $1^1/_2$-inch margins on all four sides, and limited in length to two manuscript pages.

Clinical Manifestations of Brawny Scleritis

Editor:

The article, "Clinical manifestations of brawny scleritis" (Am. J. Ophthalmol. 85:781, 1978), by S. E. Feldon, J. Sigelman, D. M. Albert, and T. Smith, points out how difficult it is to diagnose scleral inflammation when it occurs posterior to the equator of the eye, and the ineffectiveness of the phosphorus uptake test in differentiating this condition from other masses in the posterior segment.

However, we would like to plead for the abandonment of the term brawny scleritis. This was a clinical description of a granulomatous form of scleral disease associated with conjunctival edema described by Schlodtmason in 1897, and, in the original descriptions never extended behind the equator. The physical signs described as brawny scleritis can be observed in many patients with early anterior necrotizing scleral disease and certainly not all patients with posterior scleritis (including those described in the article) have the edema and gross redness said to occur in brawny scleritis.

Posterior scleritis is a severe condition, which not only produces the proptosis, exudation, detachments, and masses in the posterior segment, but also macular and disk edema. If ophthalmologists are to recognize posterior scleritis, it is important that everyone adhere to a single nomenclature.

P. G. WATSON, F.R.C.S.
A. J. LYNE, F.R.C.S.
D. LLOYD-JONES, F.R.C.S.
London, England

Choroidal Folds

Editor:

In the article, "Choroidal folds" (Am. J. Ophthalmol. 86:380, 1978), by F. E. Cangemi, C. L. Trempe, and J. B. Walsh, the authors show in Figure 2 a photograph of "markedly irregular choroidal fold formation noted after a scleral buckling procedure." These irregular folds are definitely not in the choroid. They represent remaining irregularities of the outer retinal surface. During retinal detachment the retina is edematous, causing the outer retinal layers to show folds.[1-3]

When the retina is reattached, especially after drainage of subretinal fluid, these irregularities may remain visible because the retina did not have enough time to flatten out while dehydrating.

ROBERT MACHEMER, M.D.
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Peripapillary Pigmentary Retinal Degeneration

Editor:

In the article, "Peripapillary pigmentary retinal degeneration" (Am. J. Ophthalmol. 86:65, 1978), by K. G. Noble and R. E. Carr, the authors state that "in none of these four cases was there evidence on fluorescein angiography of choriocapillaris atrophy." However, if one compares Figures 2 and 6 a marked patchy delay in the appearance of the dye in the choroid beneath the lower temporal vessels is evident in areas where there are pigment clumps. This phenomenon is typically seen in areas of patchy atrophy of choriocapillaris.[1] Without having the whole angiographic sequence for interpretation it is impossible to decide whether the atrophy of choriocapillaris in the areas of delayed filling is total or partial, in no case can the choriocapillaris be normal.[2]

LEA HYVARINEN, M.D.
Helsinki, Finland

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Corneal Decomposition after Corneal Endothelium Cell Count

Editor:

Lowered endothelial cell density may lead to a higher incidence of corneal

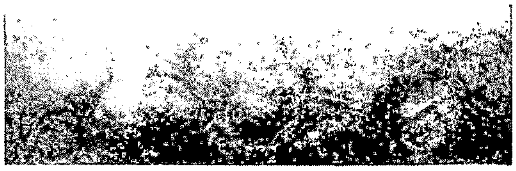

Figure (Hoffer). Endothelial cell count 18 months after lens implantation.

dystrophy. No definite limit has been set whereby one may predict decompensation.

We examined a 73-year-old woman who underwent intracapsular cataract extraction and lens implantation (Binkhorst-Federov) by means of the open-sky technique in October 1975. The patient's postoperative course was uncomplicated and seemed a complete success with a well-centered lens, a clear cornea, and a visual acuity of 6/6 (20/20). A routine endothelial cell count done in April 1977 (18 months postoperatively) resulted in 370 cells/mm^2 (Figure). The patient did well for one year and then noted a decrease in visual acuity. Examination during the next six months showed corneal decompensation and ultimately bullous keratopathy. Vision decreased to such an extent that corneal transplantation was required in 1978.

Until now no prospective endothelial cell count has been identified with ultimate corneal decompensation. This case demonstrated a cell count of 370 cells/mm^2, which required corneal transplantation one year after recording. Further studies and follow-ups should be done on patients with cell counts of less than 1,000 cells/mm^2 to document decompensation time for patients with low cell counts.

KENNETH J. HOFFER, M.D.
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Ophthalmic Miniatures

Editor:

I was most interested in an "Ophthalmic Miniature" (Am. J. Ophthalmol. 86: 552, 1978). This form of sun-induced sneeze reflex has been investigated in some detail by an afflicted physician-geneticist (M. H. K. Sokeir) and published by Dr. Shokeir and colleagues in the National Foundation-March of Dimes Birth Defects: Original Article Series 14 (6B): 361-363, 1978. The article is entitled "THE ACHOO syndrome (autosomal dominant compelling helio-ophathalmic outburst syndrome)."

GLENN C. SZALAY, M.D.
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OPHTHALMIC MINIATURE

My boats, the usual small dug-out for myself and the larger one for kit and servants, drew up opposite the company's compound. The Forest Manager saw me down to the bank.

"Want to see me sneeze?" he queried suddenly.

What does a junior forest assistant say to his boss when asked such a question? Since I was his junior, I replied in the affirmative, whereupon he glanced quickly up at the cloudless heavens and then let forth a sternutation that sent the ready plover up in clouds above the trees on the opposite bank.

"I can always sneeze when I look at the sun," he said with satisfaction.

Reginald Campbell, *Teak Wallah*
London, Hodder & Stoughton, 1935

Cystoid Macular Edema After Cryotherapy

Editor:

In the article, "Cystoid macular edema after cryotherapy" (Am. J. Ophthalmol. 86:572, 1978), by R. Kimball, P. Morse, and W. Benson, the authors describe a case of cystoid macular edema occurring after cryotherapy of peripheral retinal holes. They state that "because cystoid macular edema has never been reported as accompanying untreated retinal breaks, it seems that in this case, it was caused by the cryotherapy."

I disagree with this statement. I have personally seen cystoid macular edema with untreated retinal holes, and this has also been noted with untreated peripheral retinal holes by Wise, Dollery, and Henkind in their text, "Retinal Circulation." Moreover, they associated successful repair of the retinal holes with the disappearance of the microcystic macular lesions and full functional recovery.

But even if cystoid macular edema had not been previously observed with untreated peripheral retinal holes, I believe that in a solitary case report which demonstrates a temporal relationship, no conclusive statement can or should be made concerning causality. At best, one could say that there is a possible association of cystoid macular edema with either peripheral holes or with cryotherapy of peripheral retinal holes, or both. It is most important to challenge the authors' definitive statement concerning cryotherapy in this case report, especially now with increasing patient awareness and the desire for precise information about current therapeutic modalities.

MURRAY A. WOLKSTEIN, M.D.
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Reply

Editor:

We thank Dr. Wolkstein for expressing his doubts that the cryotherapy caused cystoid macular edema. However, we believe that, unless known or proven to be otherwise, the physician should accept the knowledge that complications are a result of his treatment. In our patient, the short interval between our treatment and the development of cystoid macular edema, together with the decrease in visual acuity, strongly suggests a cause and effect relationship. We think it important to cite Ryan's study[1] of cystoid macular edema occurring after cryotherapy and scleral buckling as a precedent to reporting the association of cystoid macular edema with cryotherapy alone.

Furthermore, Dr. Wolkstein stated that he has seen cystoid macular edema associated with peripheral retinal breaks and that cystoid macular edema has even responded to treatment of the breaks. However, in reviewing the most current reports,[2-4] it seems that the association of peripheral retinal breaks and cystoid macular edema is so rare as to be unmentionable. Nevertheless, Dr. Wolkstein discusses longstanding holes, not short term breaks, and fluorescein angiography was not performed. If this association is common, perhaps it should be formally studied with intravenous fluorescein and reported.

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PETER H. MORSE, M.D.
WILLIAM E. BENSON, M.D.
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Vitrectomy and Wagner's Vitreoretinal Degeneration

Editor:

The patients described by G. C. Brown and W. Tasman in their article, "Vitrectomy and Wagner's vitreoretinal degeneration" (Am. J. Ophthalmol. 86:485, 1978), as most if not all of those described in previous reports, have a heritable disorder which is clearly separable and distinct from that originally described by Wagner.[1] Hence, the terms vitreoretinal degeneration of Wagner and Wagner disease as used in the Brown and Tasman article, as well as in previous articles are generally inaccurate and may result in inadequate case evaluation.

In 1938, Wagner[1] described what he

referred to as hereditary hyaloideoretinal degeneration in 13 patients from a three-generation, 51-member pedigree. In 1960, Bohringer, Dieterle, and Landolt[2] reported their follow-up findings on the family initially described by Wagner (Wagner family), by then in its fourth generation. Eighteen new patients were studied, ten of whom were affected. Finally, Ricci[3] published his observations on three of the patients originally described by Wagner and five of the "new" patients described by Bohringer, Dieterle, and Landolt.[2]

These three reports[1-3] make the following clear. The disease described by Wager is not characterized by severe myopia. Rather, as emphasized by Wagner, the patients " . . . almost entirely (have) a very low degree of myopia." Thus, all but three of his patients were no more than a −4.00 in their more myopic eye. Although vitreous liquefaction, peripheral preretinal membranes, and radial perivascular pigment changes were characteristic, no retinal detachments were reported in any of the original Wagner family patients. Further, if not by ages 6 to 9 years, then by puberty, regular point-like anterior and posterior cortical opacities appeared in the crystalline lenses of the affected. These opacities gradually increased in size as well as number and spread into the nucleus, causing a significant visual acuity decrease by the third to fourth decades, if not sooner. A decreased electroretinogram and optic atrophy were less emphasized features. No systemic aberrations were mentioned in any of the patients.

Although the vitreoretinal changes in Brown and Tasman's patients, and in those with what is often referred to as vitreoretinal degeneration of Wagner are similar to those present in the Wagner family, there are several other features that distinguish the Wagner family from those described in subsequent reports. As noted, none of the affected in the Wagner family were described as having developed retinal detachment, whereas the three patients of Brown and Tasman had a range from −4.00 to −16.00, in contrast to the mild myopia stressed by Wagner. Severe myopia (greater than −4.00 to −6.00) is usually observed in patients described as being examples of Wagner disease. Further, the type and progression of crystalline lens opacities emphasized in the Wagner family are seldom observed in other so-called Wagner patients.

Whether the patients described by Brown and Tasman, like most other examples of hereditary hyaloideoretinal degeneration of Wagner are patients with the Stickler syndrome,[4,5] or some heterogenetic variant thereof remains uncertain. Certainly, severe myopia, nontraumatic retinal detachments, often in the first two decades of life, and vitreoretinal changes like those described by Wagner are characteristic of the Stickler syndrome.

Thus, it is historically incorrect to use the term hereditary hyaloideoretinal degeneration of Wagner or some other eponymic variation for patients with severe myopia, retinal detachment, and characteristic vitreoretinal degeneration. More importantly though, these terms obscure the need for a thorough systemic examination of such patients to look for other signs of the Stickler syndrome or some other as yet to be defined entity. Additional features of the Stickler syndrome include arthropathy (85%) secondary to spondyloepiphyseal dysplasia, cleft palate (28%) which may be just a submucous cleft and therefore clinically appreciable only by palpation, micrognathia (17%), and neurosensory deafness (9%).[5] The need for a complete systemic evaluation cannot be overemphasized as this is the only means to determine if such patients have the Stickler syndrome or, if as is more likely, there is additional genetic heterogeneity in this group.

In patient management an accurate diagnosis is mandatory for appropriate treatment, genetic counseling, useful referral consultations, and provision of a reliable prognostication. What is now usually called vitreoretinal degeneration of Wagner is one of perhaps several entities clearly separable and distinct from the condition originally described by Wagner. Awareness of the genetic heterogeneity in patients with hyaloideoretinal degeneration will permit accumulation of the data necessary to permit assortment of these similar yet genetically separate entities.

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Reply

Editor:

Although nine of Wagner's original 13 patients had myopia of less than or equal to −4.0 diopters, four, or approximately one third, had myopia ranging from −5.25 to −30 diopters in the more myopic eye. Wagner's paper states, " . . . without exception, all affected persons exhibited myopia, usually low grade, but sometimes more severe." Thus the presence of severe myopia in itself does not seem to exclude our patients and those of other investigators[2,3] from being Wagner's variants.

Descriptions of cataract are varied in Wagner's original article. Included are anterior and posterior cortical punctate and stellate opacities, subcapsular opacities, and diffuse homogenous nuclear turbidity. One of our patients had a posterior subcapsular lens opacity, whereas two did not. Two of Wagner's juvenile patients (ages 5 and 6 years) had clear lenses, and cataracts were not usually prominent until adolescence. Certainly other authors[2-4] have noted these opacities, which are frequently cortical, but may vary.

Wagner did not originally describe the association of retinal detachment in his patients, a point we made in our article. Jansen,[4] though, noted the frequent occurrence of retinal detachment, often at an early age, in his patients with mild to moderate (although sometimes severe) myopia, cataracts, optically empty vitreous with membranes, and peripheral and perivascular retinal pigmentation. His descriptions are similar to those of Wagner and emphasize the association with retinal detachment.

Palatoschisis has been well noted in Wagner's variants,[2,5] as have hyperextensible joints and chondrodysplasia.[2] Micrognathia and glassoptosis in two patients with Wagner's disease and Pierre Robin syndrome have also been described.[2] It was not our objective to discuss the systemic associations known to occur with Wagner variants but rather to suggest therapy for recurrent retinal detachment in the disease.

When one considers that most diseases involve a continuum along the spectrum of manifestations, it is not unreasonable to group the original Wagner patients and subsequently described patients.[2-5] Certain points of selected articles can usually be found to support disassociation, as well as association, of patient groups. Whether Wagner's disease and Stickler's syndrome, described 27 years later at a time of greater medical sophistication, are distinctly separate clinical entities or variants of the same genetic entity is a difficult question to resolve. It is bold to say with certainty that they are clearly separable and distinct.

In summary, whether one chooses to label the patients we have described as having Wagner's disease or Stickler's syndrome, both points of view can probably be well supported. Of more importance is the hazard of eponymic terminology where heredogenetic variation tends to occur. Indeed, in retrospect some of our patients probably do more fit Stickler's description,[6] but this should not divert the reader from the knowledge that patients with severe myopia and a malignant form of perivascular and equatorial lattice degeneration associated with an optically empty vitreous cavity and membrane formation may benefit from vitrectomy if scleral buckling fails. As has Dr. Judison in his letter, we wish to emphasize again the wide variability of hereditary disease.

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WILLIAM TASMAN, M.D.
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BOOK REVIEWS

Ocular Pharmacology. By William H. Havener. St. Louis, C. V. Mosby Co., 1978. Hardcover, index, table of contents, 377 black and white figures. $45

Ocular pharmacology has grown in leaps and bounds, especially during the past 12 years. This text of ocular pharmacology by Havener is now in its fourth edition. The first one appeared in 1966. This fourth edition contains comprehensive references of the past 20 years, particularly with respect to the use of ophthalmic medications and the responses of eye diseases to such treatments. The author continues to review and classify with clarity. Prejudices that have developed because of his own considerable clinical experience do induce some bias into the presentation; however, at all times the author makes his preferences known.

This is a monumental accumulation of data and references, well organized, easy to read, and understand. The contents are included under the same 38 different chapter headings as in the third edition.

Each chapter contains some new information and there has been a serious attempt to keep all of the material up to date. This is impossible in any text. For example, this one has no significant mention of the role of beta blocking agents on ocular functions particularly on intraocular pressure or glaucoma. The section on drugs useful in fungus endophthalmitis is limited to a discussion of amphotericin. Although triflurthymidine is considered in herpetic ocular infections, there is no mention of the cycloquanines. These are not oversights. They simply demonstrate the difficulties and roadblocks encountered with publishing deadlines.

Ocular pharmacology is not only a science but also an art and Dr. Havener is thoughtful, articulate, and lucid in dealing with the placebo effects, patient compliance, and the individuality of patient response.

The volume puts together information on the eye that toxicologists or pharmacologists would otherwise have to search out from many different sources. It covers effects observed in both humans and experimental animals. The fundamental mechanisms are considered as well as the clinical applications.

Most of the drugs discussed here are properly those available in the United States; however, new drug introductions in countries such as England differ from those in the United States. Approximately 2.5 times as many became available first in England, then in the United States during the years of 1972 to 1976. Ophthalmology also differs in this regard, not as much perhaps as with cardiovascular, peptic ulcer, and central nervous system drugs. It might be stimulating for readers to have available information concerning drugs clinically available in other countries for ocular therapy, for example, clonidine and bupranolol, both of which are available in Germany; pindolol, chromolyn, and butazolidine, which although available in this country for systemic use, are not available for ocular use as they are in England.

The real strength of this book is its value to all levels of individuals engaged in pursuits associated with the action of pharmacologic agents on ocular tissues and functions. This is the result of its careful documentation of the pharmacologic publications dealing with experimental studies in vitro and in vivo in experimental animals and in clinical practice.

The volume maintains the tradition of excellence begun by William H. Havener in 1966. There is no hesitance on my part in recommending this book for students, residents, investigators, and clinicians.

IRVING H. LEOPOLD

Soft Contact Lenses. Clinical and Applied Technology. By Montague Ruben. New Jersey, John Wiley & Sons, 1978. Hardcover, 496 pages, index, table of contents, 228 black and white figures, 56 color plates. $29

Since the Czechoslovakian patent for soft lenses was submitted in 1962 for the original soft (HEMA) lens, 17 different types of soft lenses have been approved for use in the United States. Numerous other experimental lenses are now in various stages of planning and production. This book presents the entire spectrum of soft lens technology to the practitioner. It is certainly not a fitting guide and does not pretend to be so. It should be invaluable, however, to the novice practitioner of soft contact lens fitting as a reference source for lens handling, lens hygiene, and lens interaction with various medications, as well as the assessment of hydrogel lens factors. This book would certainly serve the experienced lens fitter as a complete reference source for the various physical properties of lenses.

The book begins with the historical development of hydrogel material and devotes considerable attention to the applied sciences of chemistry, physiology, and optics as they relate to soft lens materials. Some of the sections on physical properties and gas transmission in soft lens systems may be beyond the interest of the average practitioner, but they serve as background for understanding the basic differences in lens material as well as "why lenses with similar names are not." The sections on clinical practice deal with lens design and guidelines for fitting soft lenses, for cosmetic, aphakic, and extended wear situations. Toric soft contact lenses, keratoconus lenses, bandage lenses, and lenses as drug delivery systems are also discussed. Specific fitting details are not emphasized, but rather a broad overview of the science of clinical practice is presented.

The author presents an in depth review of the complications of soft lens wear from a chemical, physical, and biologic standpoint. The science of soft lens hygiene is taken from basic chemistry to clinical practice.

In the final section on the technology of manufacture, the reader follows various manufacturers of soft lenses through their stages of lens production. Manufacturers' quality control is described and methods of verification of manufacturers' specifications usable in the practitioner's office are also given.

KEITH S. MORGAN
HERBERT E. KAUFMAN

SYMPOSIA

Modern Problems in Ophthalmology, Vol. 19. Colour Vision Deficiencies IV.

Edited by G. Verriest. Fourth Symposium of the International Research Group on Colour Vision Deficiencies, Parma, 1977. Basel, S. Karger AG, 1978. Hardcover, 348 pages, index, 146 black and white figures, 37 tables. $129.25

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Birch-Cox, J. (*London*): **Defective colour vision in diabetic retinopathy before and after laser photocoagulation**

Kalmus, H. (*London*): **Telenomy of normal and defective colour vision**

Cobb, S. R. (*Glasgow*): **Examination of class I, class II, normals and colour defectives on exchange thresholds colorimeter and analytical anomaloscope**

Ruddock, K. H., and Waterfield, V. A. (*London*): **Central representation of colour vision deduced from studies on a subject with a central colour vision defect**

van den Berg, T. J. T. P. (*Amsterdam*): **Rod-cone interaction with homogenous field stimulation**

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FRANK W. NEWELL, M.D.
Editor-in-Chief
AMERICAN JOURNAL OF OPHTHALMOLOGY

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

American Journal of Human Genetics

HEREDITARY RETINOBLASTOMA: DELAYED MUTATION OR HOST RESISTANCE? Matsunaga, E. (Dept. Human Genetics, National Inst. Genetics, Shizuoka-Ken, Japan). Am. J. Hum. Genet. 30:406, 1978.

There are generally two forms of retinoblastoma, hereditary and nonhereditary or sporadic. The sporadic form is thought to represent about 60% of all retinoblastomas while the remaining 40% are hereditary which can be inheredited from a parent who survived the disease, an unaffected parent carrying the gene or it can be due to a new dominant mutation in the gonads of a healthy parent. One of the variables that exists in hereditary retinoblastomas is degree of penetrance and there are two theories to explain the variation. One states that the penetrance and expressivity in the gene carrier is a variable determined by genetic and environmental factors. While another theory postulates delayed mutation which occurs primarily due to exposure to chemical mutagens. The data in this paper suggests that it is the first explanation that is true and there appears to be little need to postulate delayed mutation for the retinoblastoma locus. (10 tables, 36 references)—David Shoch

Archives of Ophthalmology

INTENTIONAL SURGICAL OVERCORRECTION OF ACQUIRED ESOTROPIA. Dankner, S. R., Mash, A. J., and Jampolsky, A. (Smith-Kettlewell Inst. Visual Sci., San Francisco, Calif.). Arch. Ophthalmol. 96:1848, 1978.

Fifty-eight patients with surgically treated acquired nonaccommodative esotropia were studied retrospectively, to ascertain what immediate surgical result gives the best chance for a postoperative bifoveal fusional result. Of the cases that were initially overcorrected (exodeviations), approximately two thirds were fusing six months or more after surgery. Of the cases that were initially aligned (orthophoric) or undercorrected (esodeviations), only one third were fusing after the same period. These results suggest that intentional surgical overcorrection of cases of acquired nonaccommodative esotropia that have a good fusional potential (i.e., equal vision) does appear to be a desirable goal for the immediate postoperative period. (4 tables, 17 references)—Authors' abstract

OSSEOUS CHORISTOMA OF THE CHOROID SIMULATING A CHOROIDAL MELANOMA: ASSOCIATION WITH A POSITIVE ^{32}P TEST. Williams, A. T., Font, R. L., Van Dyk, H. J. L., and Riekhof, F. T. (Div. Ophthalmol., Univ. Utah, College Med., Salt Lake City, Utah). Arch. Ophthalmol. 96:1874, 1978.

The authors report the clinical and histopathologic features of an osseous choristoma of the choroid that appeared in a 26-year-old woman. Her diagnostic evaluation included a radioactive phosphorus (^{32}P) uptake test, which was 270% positive and led them to believe that the lesion was an atypical malignant melanoma. The extent of true ossification within this tumor, seen microscopically, explains the false-positive uptake of radioactive phosphorus by this benign tumor; it is well known that bone avidly accumulates radioactive phosphorus. The authors recommend that all "atypical choroidal melanomas" be studied for the presence of bone, with bone-free roentgenograms, ultrasonography, and possibly, computerized tomographic scans before ^{32}P testing or enucleation. This is apparently the second histopathologically proved case of an osseous choristoma of the choroid to be reported in the literature. (6 figures, 6 references)—Authors' abstract

ENDOTHELIAL CELL POPULATION CHANGES OF HUMAN CORNEA DURING LIFE. Laule, A., Cable, M. K., Hoffman, C. E., and Hanna, C. (Depts. Ophthalmol. and Pharmacol., Univ. Arkansas Med. Sci., Little Rock, Ark.). Arch. Ophthalmol. 96:2031, 1978.

A photo slit lamp was used to obtain color, specular reflex, high magnification photographs of the corneal endothelium of subjects ranging in age from three to 88 years. Multiple areas of the cornea were examined to determine the endothelial cell population. No appreciable difference in cell density was found between the right and left eyes of the subjects nor between male and female subjects of similar age. Apparent defects in the endothelial cell coverage of Descemet's membrane were found in subjects as young as 20 years of age and with increased frequency in older age groups. These defects were at times associated with variations in endothelial cell populations between the central and peripheral cornea. The average corneal cell population fell from nearly one million cells in the first years of life to about one third that number by the eighth decade of life. (8 figures, 1 table, 11 references)—Authors' abstract

THE MECHANISM OF TIMOLOL IN LOWERING INTRAOCULAR PRESSURE IN THE NORMAL EYE. Coakes, R. L., and Brubaker, R. F. (Dept. Ophthalmol., Mayo Clinic, Rochester, Minn.). Arch. Ophthalmol. 96:2045, 1978.

A single-drop, double-masked, randomized, placebo-controlled study of the mechanism of the ocular-pressure lowering property of timolol maleate, a β-adrenergic blocker, was carried out in 23 normal subjects, using fluorophotometry. Timolol suppressed aqueous formation in all subjects. The range of suppression was 13% to 48%, with a mean ± SD of 34% ± 9%. The drug had no effect on anterior chamber volume or endothelial permeability to fluorescein and, apparently, had no effect on outflow resistance. No differences were observed between its effect on men and women or between eyes with light and dark irides. (2 tables, 5 references)—Authors' abstract

Brain

FACTORS INFLUENCING THE RISK OF MULTIPLE SCLEROSIS DEVELOPING IN PATIENTS WITH OPTIC NEURITIS. Compston, D. A. S., Batchelor, J. R., Earl, C. J., and McDonald, W. I. (Moorfields Eye Hosp. and Inst. Neurol., Queen Square, London). Brain 101:495, 1978.

One-hundred and forty-six patients who had presented with optic neuritis but without evidence of demyelination elsewhere in the nervous system, and in whom no specific cause could be identified, were reassessed clinically between one month and 23 years after the onset. Fifty-eight patients (40%) had developed MS. All 146 patients were HLA-typed. Three factors were identified which were significantly associated with the development of MS: positive typing for the HLA antigen BT 101, winter onset of the initial attack of optic neuritis in BT 101-positive patients only, and recurrent attacks of optic neuritis. The application of these results to the individual patient is of limited use. However, recurrent attacks of optic neuritis should be given the same significance in the clinical classification of MS as episodes of demyelination occurring elsewhere in the central nervous system in a patient with a previous attack of optic neuritis. The results suggest that optic neuritis is caused by two different environmental agents or groups of agents and that the agent which is most common in the winter leads to the development of MS in the genetically susceptible individual. The agent more common in the summer is much less likely to cause MS in either susceptible or non-susceptible individuals. It is suggested that the presence of the HLA antigens associated with MS confers a specific disadvantage on individuals in the ability to handle infection by the MS causative agent and that this allows damaging immunological processes to develop. (1 figure, 5 tables, 54 references)—Authors' abstract

British Journal of Ophthalmology

EFFECTS OF VARIOUS CONCENTRATIONS OF DIETHYLCARBAMAZINE CITRATE APPLIED AS EYE DROPS IN OCULAR ONCHOCERCIASIS, AND THE POSSIBILITIES OF IMPROVED THERAPY FROM CONTINUOUS NON-PULSED DELIVERY. Jones, B. R., Anderson, J., and Fuglsang, H. (Dept. Clin. Ophthalmol., Moorfields Eye Hosp., London). Br. J. Ophthalmol. 62:428, 1978.

Although diethylcarbamazine (DEC) is the most effective microfilaricide available for treating ocular and other forms of onchocerciasis, the oral use of the drug in cases of severe ocular involvement is fraught with the danger of serious adverse host reactions to the disintegrating microfilariae. The present paper is concerned with a search for DEC-concentrations and delivery schedules which are topically effective without eliciting serious damage to ocular tissues. Close biomicroscopic observations on 14 patient-volunteers have enabled the authors to determine three drug delivery rates (calculated from the drug concentration and the number of drops administered per hour) which correlate fairly well with three types or degrees of host responses. Minimum DEC-delivery rates of 0.1 μg/hr were observed to cause increased entry of microfilariae into the cornea, followed by signs of death and disintegration of the larvae without appreciable inflammatory host response; delivery rates of 0.6 μg/hr brought adverse reactions in the form of globular limbal infiltrates with itching and congestion to the fore; at delivery rates of 1.0 μg/hr the adverse reactions were more severe and included signs of vasculitis in limbal and corneal blood vessels. Still higher doses elicited acute anterior uveitis with flare, cells and even hypopyon in the anterior chamber. The authors reason that the margin between delivery rates causing death of the organisms and the rates causing untoward inflammatory reactions is probably sufficient to allow delivery of DEC by a continuous (non-pulsed) method of administration which may achieve substantial clearing of the microfilarial load without adverse reactions. For this form of topical administration to the eye the authors are considering a modification of the Ocusert, the "Supratarsal Delivery Platform," which was originally designed for mass chemotherapy of trachoma. After preliminary clearing of the microfilarial load by carefully controlled delivery of DEC, it may be possible to continue the treatment by less strictly controlled delivery rates of DEC (in medicated cooking salt), or even attack the adult worms with the trypanocidal agent suramin which, without preparatory microfilaricidal therapy, has proved to be highly toxic. (6 figures, 4 tables, 25 references)—Peter C. Kronfeld

ASPECTS OF CORNEAL CHANGES IN ONCHOCERCIASIS. Tønjum, A. M., and Thylefors, B. (Univ. Eye Dept., Rikshospitalet, Oslo, Norway). Br. J. Ophthalmol. 62:462, 1978.

The presence of microfilariae of *Onchocerca volvolus* (MFS) in the cornea of patients with onchocerciasis has been known since the early 1940s. Corneae containing more than 500 living larvae are not unusual in heavily infected persons. The present authors have studied the distribution pattern of microfilariae and of the opacities following their death (onchocercal punctate opacities) in the corneas of 160 patients from the Volta River Basin in West Africa. The density of microfilaria and the opacities was found to be greatest in the periphery of the nasal and temporal quadrants of the cornea, decreasing markedly toward its center. On the basis of these findings the authors regard the corium of the adjacent skin as the most likely route of entry of the MFS into the cornea. (2 figures, 3 tables, 7 references)—Peter C. Kronfeld

British Medical Journal

PUPILLARY SIGNS IN DIABETIC AUTONOMIC NEUROPATHY. Smith, S. E., Smith, S. A., Brown, P. M., Fox, C., and Sönksen, P. H. (St. Thomas's Hosp. Med. School, London). Br. Med. J. 2: 924, 1978.

Pupillary function was investigated in 36 insulin-dependent diabetics and 36 controls matched for age and sex. About half of the diabetics had evidence of peripheral somatic or autonomic neuropathy, or both. The diabetic patients had abnormally small pupil diameters in the dark and less fluctuation in pupil size (hippus) during continuous illumination than the controls. They also had reduced reflex responses to light flashes of an intensity adjusted for individual retinal sensitivities. The pupillary findings were compared with results of five tests of cardiovascular function and five tests of peripheral sensory and motor nerve function. Almost all the patients with autonomic neuropathy had pupillary signs, which we therefore conclude are a common manifestation of diabetic autonomic neuropathy. (3 figures, 1 table, 17 references)—Authors' abstract

Canadian Journal of Ophthalmology

GENETIC BACKGROUND OF FUCHS' HETEROCHROMIC CYCLITIS. Saari, M., Vuorre, I., Tiilikainen, A., and Algvere, P. (Depts. Ophthalmol. and Med. Microbiol., Univ. Oulu, Oulu, Finland). Can. J. Ophthalmol. 13:240, 1978.

The authors studied the genetic background of 24 patients with Fuchs' heterochromic cyclitis. Two families each had two cases of Fuchs' cyclitis in the same family; in addition an ancestor in the second family possibly had the disease as well; in both families one healthy member had simple heterochromia. One patient with Fuchs' cyclitis had congenital uveal coloboma, one pigmentary retinal dystrophy, and four had cysts of the ciliary body. The frequencies of all HLA antigens in patients with Fuchs' cyclitis compared well with the frequencies in the controls. In a family in which HLA haplotypes could be derived, the patients with Fuchs' heterochromic cyclitis showed different HLA haplotypes. The authors conclude that Fuchs' heterochromic cyclitis has a hereditary basis but its immunological component is not genetically associated with the HLA system. (3 figures, 2 tables, 20 references)—Authors' abstract

SYSTEMIC REACTION TO SUBCONJUNCTIVAL PHENYLEPHRINE. Miller, S. A., and Mieler, W. F. (Dept. Ophthalmol., Univ. Wisconsin, Madison, Wis.). Can. J. Ophthalmol. 13:291, 1978.

After a subconjunctival injection of phenylephrine, cocaine and atropine, a patient's blood pressure rose to 220/180 mmHg. This rise was followed by hypotension, pulmonary edema, and subendocardial ischemia, all of which resolved spontaneously within 12 hours. This report reemphasizes the hazardous effects on blood pressure of phenylephrine applied to the eye. (13 references)—Authors' abstract

Journal of the American Medical Association

TRANSIENT OCULOMOTOR NERVE PALSY ASSOCIATION WITH THIAZIDE-INDUCED GLUCOSE INTOLERANCE. Miller, N. R., and Moses, H. (Wilmer Ophthalmol. Inst., Johns Hopkins Hosp., Baltimore, Md.). J. A. M. A. 240:1887, 1978.

The etiology of oculomotor nerve palsy occurring in diabetes is unknown but is thought by some to be simply coincidental or in any case part of a vascular disease unrelated to the hyperglycemia. In the patient described here, glucose intolerance developed as a result of chlorothiazide ingestion. On stopping the drug the glucose intolerance disappeared and the third nerve palsy gradually cleared. This patient thus did not have true diabetes but only drug-induced hyperglycemia and this would imply that oculomotor palsy may be in fact related to the hyperglycemia. (2 figures, 6 references)—David Shoch

Journal of Nervous and Mental Disease

VISUAL CONVERSION REACTION IN CHILDREN AND ADULTS. Rada, R. T., Meyer, G. G., and Kellner, R. (Dept. Psychiatry, Univ. New Mexico School Med., Albuquerque, N.M.). J. Nerv. Ment. Dis. 166:580, 1978.

The diagnosis of visual conversion reaction (hysterical blindness) is usually made by the finding of fluctuating visual acuity, tubular visual fields and normal electrical responses. Generally adults have more psychopathology and conversion symptoms and have greater personal and social disability from the symptoms. In young children feelings of being threatened, usually because of strife within the family, tend to predominate. In general the condition is more amenable to treatment in the child where frequently the ophthalmologist, with an occasional psychiatric consultation, can cure the problem. In adults, the conversion reaction tends to be more deep-seated and referral to a psychiatrist is usually indicated. Further there is frequently a history of eye disease in the family or some other preoccupation with vision or the conversion reaction may be superimposed on a true organic lesion. In any case, the recovery is less rapid and the ultimate prognosis tends to be worse in adults. (55 references)—David Shoch

Klinische Monatsblaetter für Augenheilkunde

ANOMALIES OF COLOR VISION OF WOMEN AS AN INDICATION OF TESTICULAR

FEMINIZATION. Geisşendoerfer, T., and Ober, M. (Univ. Eye Clinic, Erlangen-Nurnberg, W. Germany). Klin. Monatsbl. Augenheilkd. 173:256, 1978.

Patients with the syndrome of testicular feminization have normal male chromosomes but are phenotypically female. There are normal female external genitalia and undescended testes. Ophthalmological examinations were done on 17 of these patients. Four patients showed defective color vision (two had deuteranomaly and two had deuteranopia). When women show congenital anomalies of color vision, testicular feminization should be thought of as a possible diagnosis. (1 figure, 15 references) — Peter R. Egbert

Neurology

EYE MOVEMENTS IN ATAXIA-TELANGIECTASIA. Baloh, R. W., Yee, R. D., and Boder, E. (Dept. Neurol., Univ. California, Los Angeles, Calif.). Neurology 28:1099, 1978.

The spectrum of eye movement disorders in six patients with ataxia-telangiectasia at different stages of progression was assessed quantitatively by electrooculography. All patients demonstrated abnormalities of voluntary and involuntary saccades. The youngest and least involved patient had significantly increased reaction times of voluntary saccades, but normal accuracy and velocity. The other patients demonstrated increased reaction times and marked hypometria of horizontal and vertical voluntary saccades. Saccade velocity remained normal. Vestibular and optokinetic fast components (involuntary saccades) had normal amplitude and velocity but the eyes deviated tonically in the direction of the slow component. The authors conclude that patients with ataxia-telangiectasia have a defect in the initiation of voluntary and involuntary saccades in the earliest stages. These findings are distinctly different from those in other familial cerebellar atrophy syndromes. (3 figures, 2 tables, 19 references) — Authors' abstract

Ophthalmologica

RETINAL ISCHEMIA (CAPILLARY NONPERFUSION) IN DIABETIC RETINOPATHY OF PATIENTS WITH AND WITHOUT SYSTEMIC HYPERTENSION. Merin, S., Ber, I., and Ivry, M. (Dept. Ophthalmol., Hadassah Univ. Hosp., Jerusalem). Ophthalmologica 177:76, 1978.

The authors studied the fluorescein angiograms of 138 patients with diabetic retinopathy. Areas of capillary nonperfusion were found in two-thirds using a macular centered photograph. There was no statistically significant correlation between the degree of capillary nonperfusion and the level of systolic and diastolic blood pressure, the duration of diabetes, the age at onset of diabetes, the age at examination, the control of diabetes and the treatment of hypertension. The only borderline statistically significant difference was found between patients with constant hypertension when compared to patients with labile hypertension. (7 tables, 20 references) — David Shoch

Ophthalmology

HYPERORNITHINEMIA AND GYRATE ATROPHY OF THE CHOROID AND RETINA. McCulloch, J. C., Arshinoff, S. A., Marliss, E. B., and Parker, J. A. (Depts. Ophthalmol. and Med., Univ. Toronto, Ontario). Ophthalmol. 85:918, 1978.

The diagnosis of hyperornithinemia and gyrate atrophy depends upon the presence of five characteristic features: (1) typical chorioretinal lesions, (2) high myopia, (3) cataracts, (4) hyperornithinemia, and (5) autosomal recessive inheritance. In addition, from their examination of three patients the authors describe four new findings: (1) decreased whole blood glutamic acid, (2) low normal intelligence, (3) hepatic mitochondrial changes, and (4) urinary excretion of ornithine methyl ester. Investigations of amino acid metabolism in vivo are consistent with the presence of a defect in ornithine ketoacid transaminase. (5 figures, 2 tables, 19 references) — Authors' abstract

BILATERAL RETINAL ARTERY AND CHORIOCAPILLARIS OCCLUSION FOLLOWING THE INJECTION OF LONG-ACTING CORTICOSTEROID SUSPENSIONS IN COMBINATION WITH OTHER DRUGS. Wilson,

R. S., Havener, W. H., and McGrew, R. N. (Univ. Arkansas Med. Sci., Little Rock, Ark.). Ophthalmol. 85:967, 1978.

Two cases are presented that illustrate the serious ocular complications that may follow the injection in the head and neck of long-acting corticosteroids in combination with other drugs. The acute clinical picture includes (1) severe bilateral visual loss; (2) hazy sensorium, (3) conjunctival vascular occlusion with edema and hyperemia, (4) superior gaze palsy, (5) pupillary abnormalities, (6) intra-arterial emboli and infarction, (7) choroidal infarcts, and (8) retinal edema. The authors stress that long-acting corticosteroids have particles capable of occluding small blood vessels. (11 figures, 15 references) — Authors' abstract

Pediatrics

PERIORBITAL CELLULITIS IN CHILDREN. Barkin, R. M., Todd, J. K., and Amer, J. (Dept. Pediatrics, Univ. Colorado Med. Ctr., Denver, Colo.). Pediatrics 62:390, 1978.

Seventy-one cases without orbital involvement, treated between 1972 and 1976, were reviewed, to develop bacteriologic data for proper treatment. Forty percent showed radiologic evidence of sinusitis, and 28% had evidence of recent local injury. A bacteriologic cause was established in 18 (25%) cases; blood cultures were positive in eight of 55 patients, yielding *H. influenzae* (five cases), *S. pneumoniae* (two cases), and *S. aureus* (one case). Culture of a contiguous wound or aspirates of the cellulitis were positive in 11 patients. Needle aspiration was positive on culture in seven of nine procedures, and showed a similar distribution of infectious agents. *H. influenzae* was demonstrated only in children under five years of age. Because *H. influenzae*, *S. aureus*, and Group A *Streptococcus* are the common pathogens, if no other organism is found on gram stain, initial treatment should consist of ampicillin and a penicillinase-resistant penicillin. Chloramphenicol is not required for treatment of uncomplicated periorbital cellulitis unless unusual circumstances exist, or a poor response to initial therapy is noted. Therapy is administered parenterally until evidence of systemic toxicity and local inflammation are resolving, after which time medication is continued orally. Nasal decongestants and topical ophthalmic antibiotics have been suggested, but have not been documented as efficacious. (3 tables, 6 references) — Paul E. Romano

Science

PHAGOCYTOSIS OF LIGHT- AND DARK-ADAPTED ROD OUTER SEGMENTS BY CULTURED PIGMENT EPITHELIUM. Hall, M. O. (Jules Stein Eye Inst., School Med., Univ. California, Los Angeles, Calif). Science 202:526, 1978.

Pigment epithelial cells in culture retain their ability to phagocytize rod outer segments. These cells phagocytize rod outer segments isolated from light-adapted rats, or from dark-adapted rats killed after the time at which the lights would normally be turned on. However, they phagocytize far fewer rod outer segments prepared in the dark from the retinas of rats killed before the onset of the normal light cycle. Phagocytosis of dark rod outer segments is variable, but that of light outer segments is reproducible. It is postulated that the effect of light is to synchronize the chemical events that occur at the surface of the rods to prepare them for phagocytosis. These processes also occur in the dark, but more slowly and irregularly than in the light. (3 figures, 14 references) — Author's abstract

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

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For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least three months before the date of occurrence.

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UNIVERSITY OF CALIFORNIA, SAN FRANCISCO: VITRECTOMY SYMPOSIUM

The University of California, San Francisco, will present a Vitrectomy Symposium and Workshop with emphasis on anterior segment technique and instrument comparison, April 18-20, 1979, at the Hyatt Regency Hotel, San Francisco. For further information, write Extended Programs in Medical Educaiton, Room 569-U, University of California, San Francisco, CA 94143; telephone (415)666-4251.

GEORGETOWN UNIVERSITY CENTER FOR SIGHT: TWO-YEAR TRAINING PROGRAM FOR OPHTHALMIC TECHNICIANS

The Georgetown University Center for Sight will offer a two-year training program for ophthalmic technicians beginning in June 1979. There is no fee. The deadline for applications is March 30, 1979. For further information, write Candace P. Wolfe, Director, Ophthalmic Technician Program, Center for Sight, 3800 Reservoir Rd., N.W., Washington, DC 20007.

ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY: 1979 MEETINGS

The spring meeting of the Illinois Society of Ophthalmology and Otolaryngology will be held at Chateau Louise, Dundee, Illinois, April 20-22, 1979. The fall meeting will be held at the Holiday Inn, Freeport, Illinois, Sept. 14-16, 1979. For further information, write A. Reese Matteson, M.D., Secretary-Treasurer, 101 W. North St., Danville, IL 61832.

WORST'S INTRAOCULAR LENS IMPLANTATION COURSES

J. G. F. Worst has scheduled the following two-day courses on intraocular lens implantation for the year 1979: March 18 and 19, May 13 and 14, Sept. 2 and 3, and Nov. 18 and 19. For further information, write Mrs. Josine L. de Meij, Secretary, Julianalaan 11, 9751 BM Haren Gn, Holland.

GEORGETOWN UNIVERSITY MEDICAL CENTER CENTER FOR SIGHT: TEN-WEEK COURSE FOR OPHTHALMIC ASSISTANTS

The Georgetown University Medical Center for Sight will hold a ten-week course for ophthalmic assistants June 25-Aug. 31, 1979. The course hours are 9:30 AM to noon, Monday through Friday. Tuition is $250. Application deadline is May 30, 1979. For further information, write Ms. Candace P. Wolfe, C.O.T., Director, Ophthalmic Technician Program, Center for Sight, Georgetown University Medical Center, 3800 Reservoir Rd., N.W., Washington, DC 20007.

WILMER INSTITUTE: 10TH ANNIVERSARY OF THE RETINAL VASCULAR CENTER

The Wilmer Institute's Retinal Vascular Center will hold its tenth anniversary celebration at Sun Valley, Idaho, June 25-July 1, 1980. The topic is "A decade of progress in diagnosis and management of retinal vascular and macular disorders." Among the panelists scheduled to appear are the following: Alan Bird, Daniel Finkelstein, J. Donald M. Gass, Morton F. Goldberg, W. Richard Green, A. Edward Maumenee, Ronald G. Michels, and Arnall Patz. For information, write Stuart L. Fine, M.D., Wilmer Institute, Johns Hopkins Hospital, Baltimore, MD 21205.

PERSONALS

DEVRON CHAR

Devron Char has been appointed chairman of the Department of Ophthalmology at Rush Medical Center, Chicago.

A. EDWARD MAUMENEE

A. Edward Maumenee presented the Seventh Annual G. Victor Simpson Lecture Dec. 16, 1978, at Washington Hospital Center, Washington, D.C. The title of Dr. Maumenee's lecture was "Intraocular lenses."

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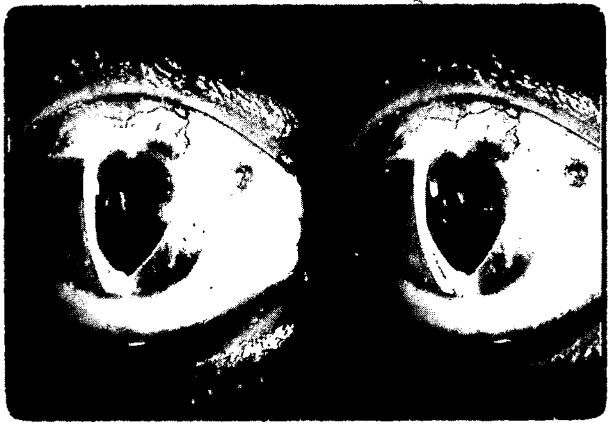

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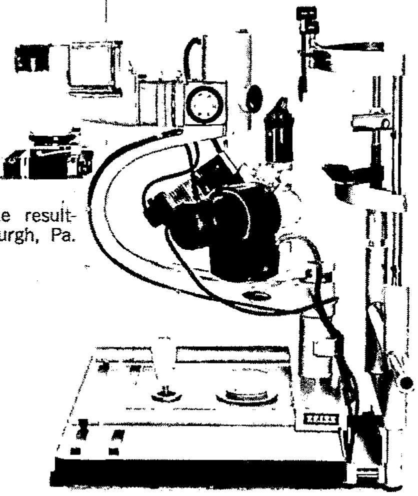

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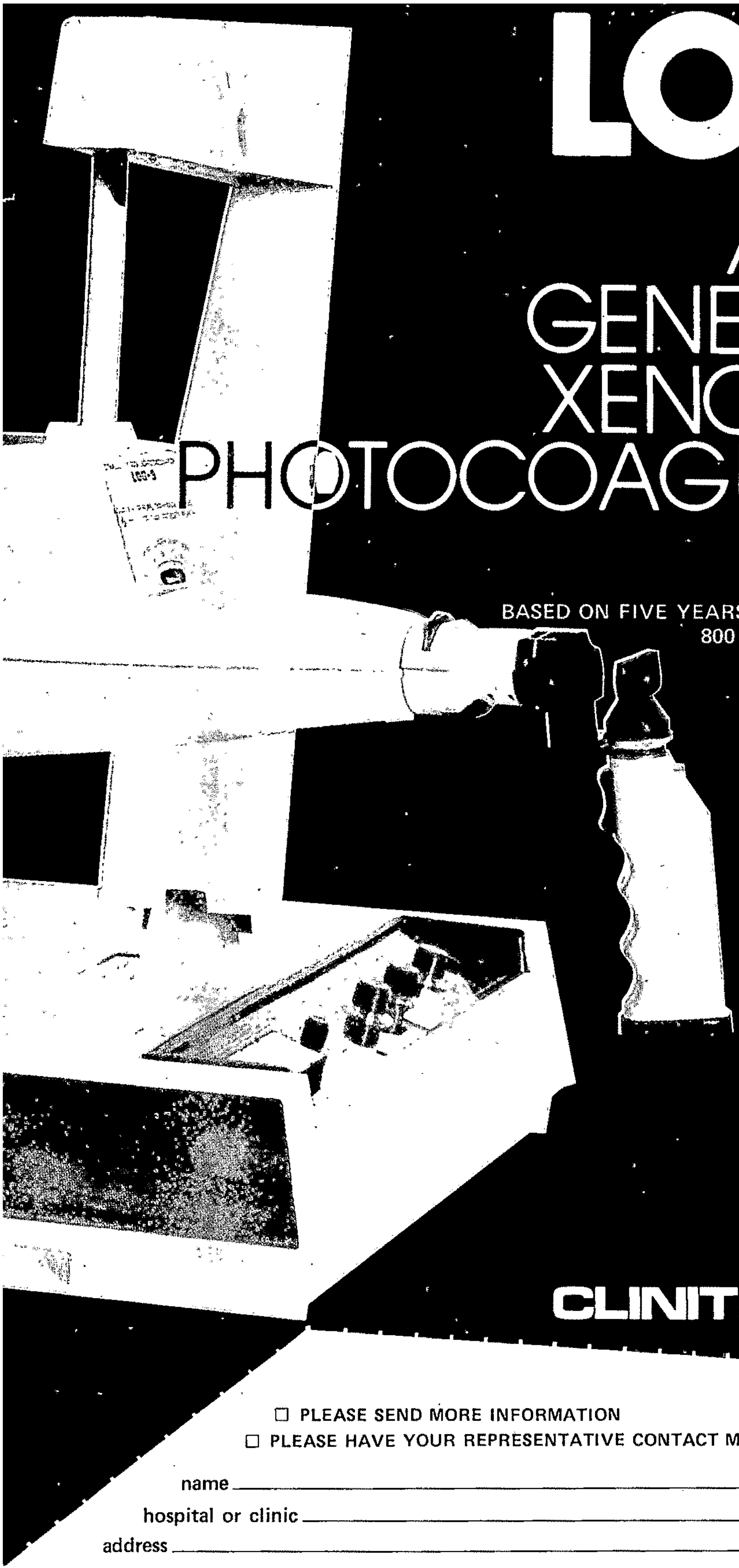

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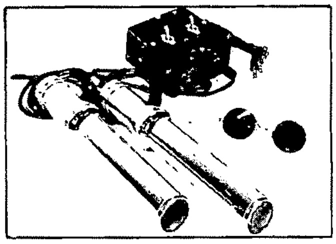

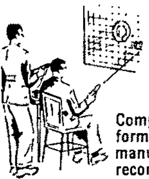

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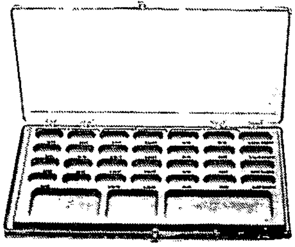

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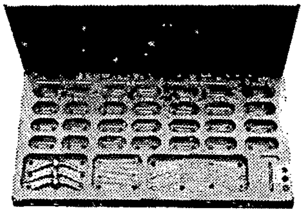

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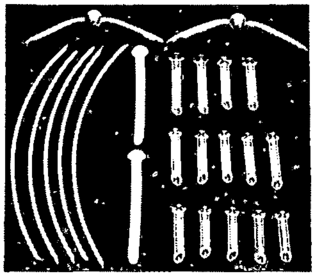

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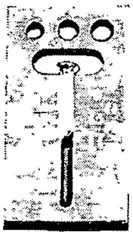

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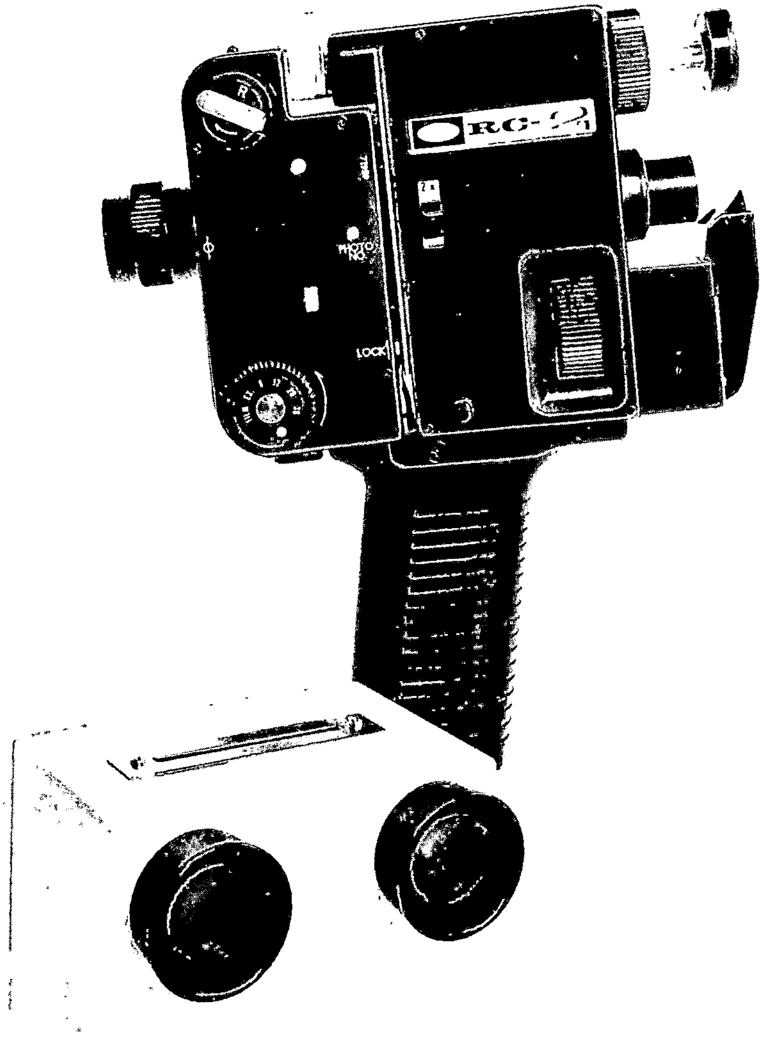

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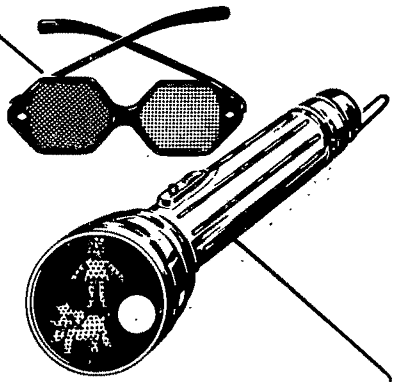

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Feb. 23, 24	Univ. of So. Calif. (USC) Estelle Doheny Eye Foundation Los Angeles, CA	Kenneth R. Diddie, M.D.	Bill Dowey (213) 226-2751
Mar. 9, 10	University of No. Carolina Chapel Hill, NC	David E. Eifrig, M.D.	Betty Nielson (919) 966-4161
Mar. 23, 24	University of Texas Medical School San Antonio, TX	David M. Hunter, M.D.	Barbara Woods (512) 696-6295
Apr. 18, 19, 20	University of California Medical School San Francisco, CA	Walter H. Stern, M.D.	Rene Vandergrift (415) 666-4251
May 11, 12	Ingalls Memorial Hospital Harvey, (Chicago) IL	David H. Orth, M.D.	Roberta Martia, R.N. (312) 333-2300, X5811
May 25, 26	Piedmont Hospital Atlanta, GA	William S. Hagler, M.D.	Karen Upchurch (404) 351-2220
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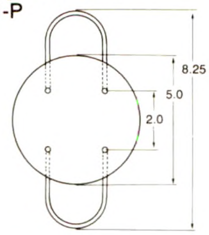

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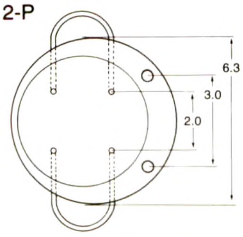

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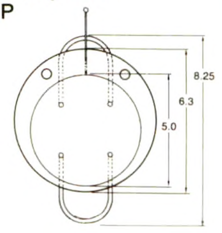

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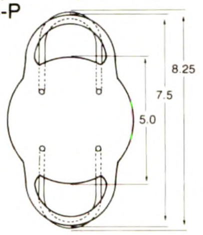

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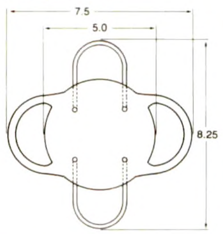

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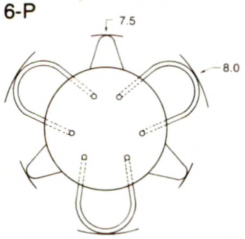

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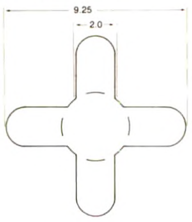

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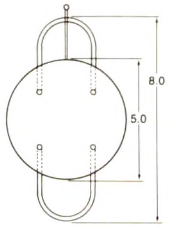

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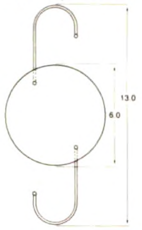

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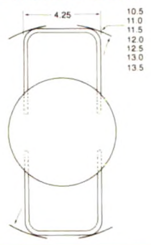

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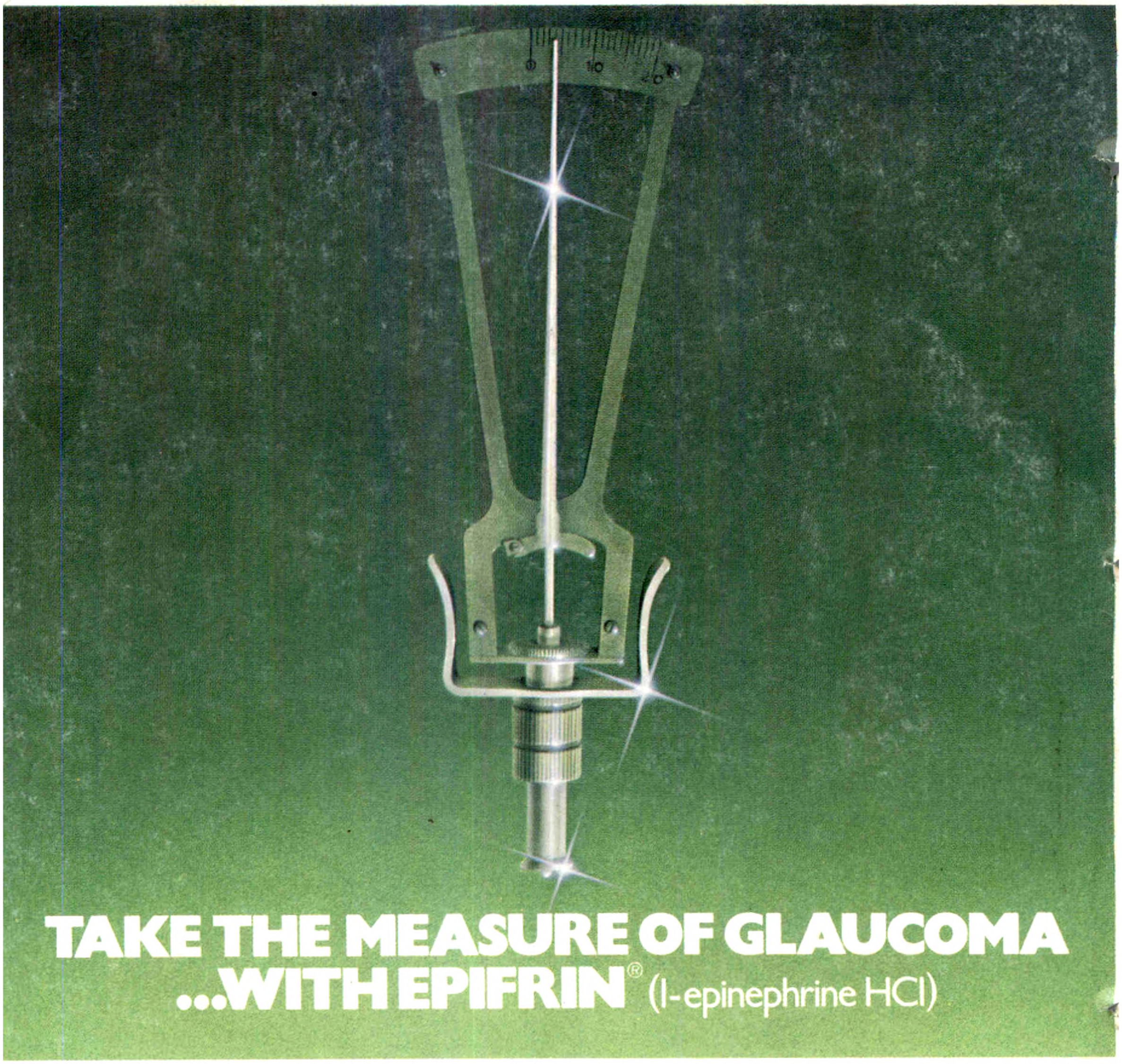
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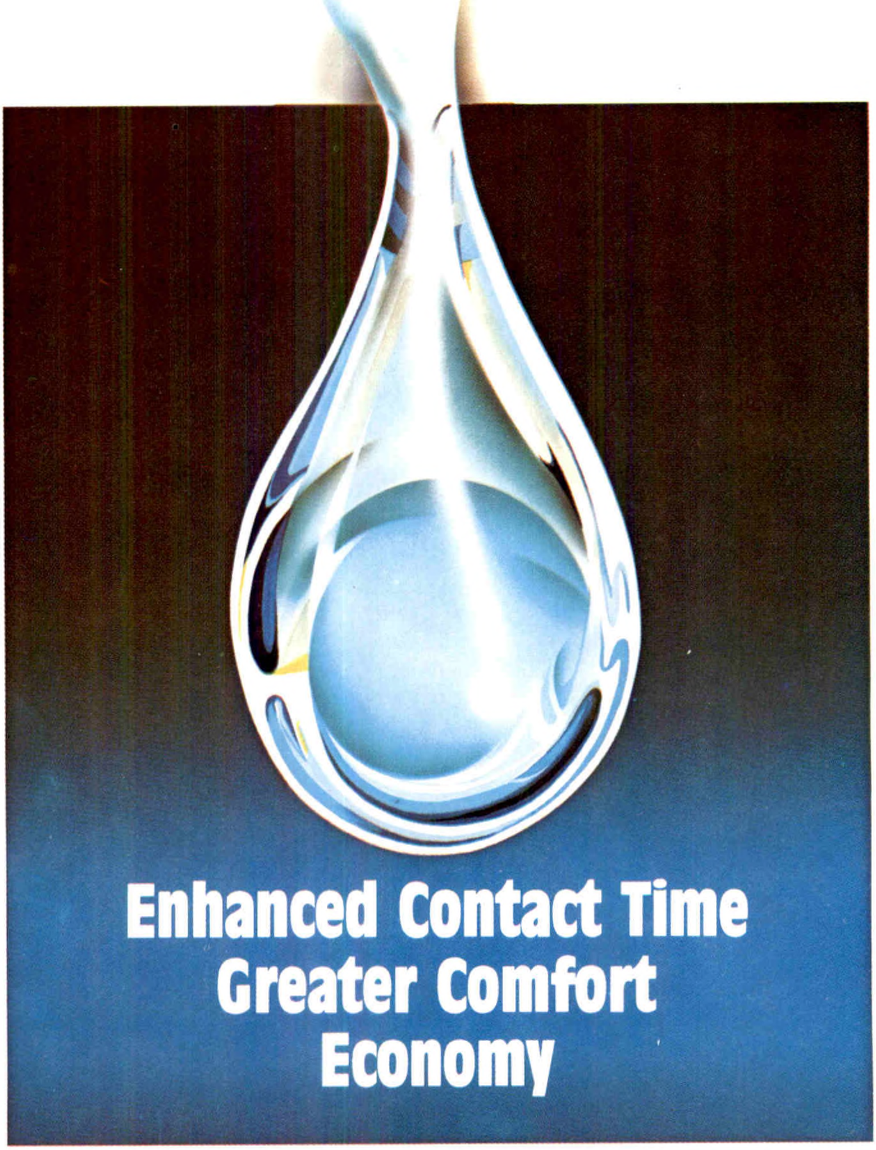

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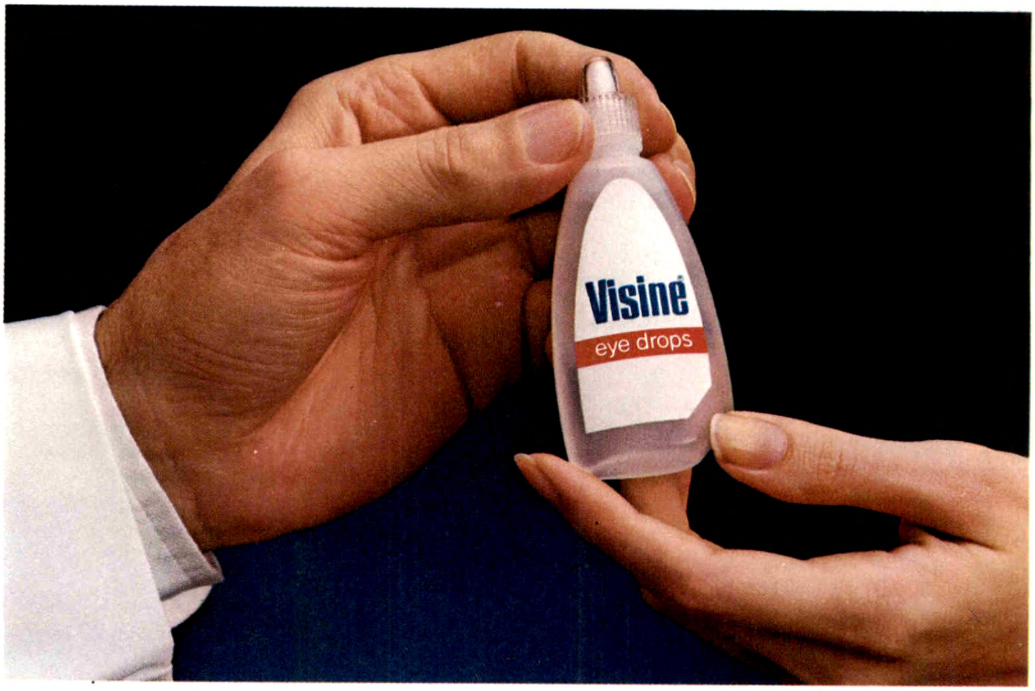

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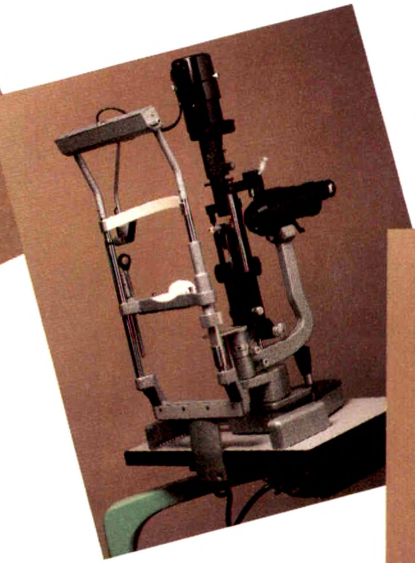

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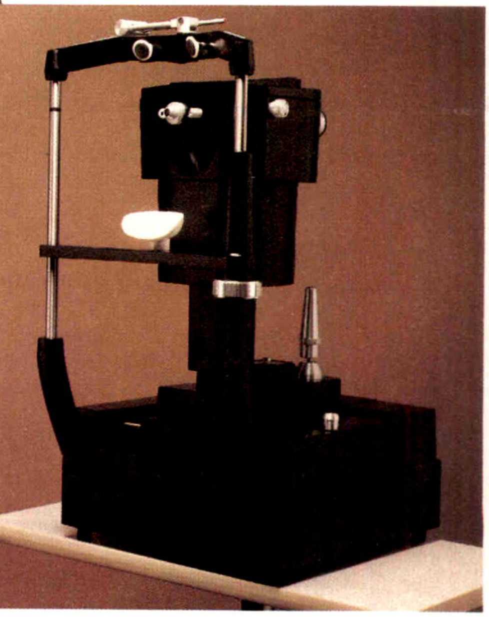

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1. Obstbaum S.A. et al, Timolol: Effect on intraocular pressure in chronic open-angle glaucoma. Ann Ophthalmol 10(10): 1347-1351, 1978.
2. Katz, I.M., Beta-blockers and the eye: An overview. Ann Ophthalmol 10(7): 847-850, 1978.
3. A study conducted by an independent research organization. Allergan Pharmaceuticals Report Series No. 148, 1979.

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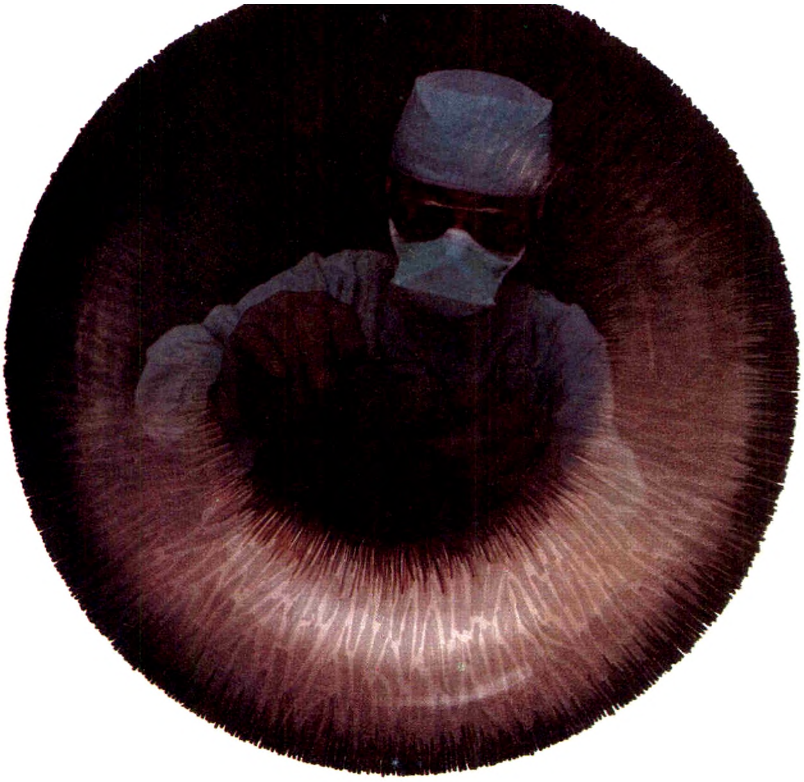

FML® (fluorometholone) 0.1% **takes the heat off postoperative inflammation.**

Inflammation following surgery can be sight-threatening. FML® (fluorometholone) helps prevent potential complications by reducing inflammation—promptly and effectively. A clinical study of almost 400 surgical procedures showed that fluorometholone significantly reduced the incidence of corneal graft rejection and post-cataract extraction complications.[1]

And what's more, FML has a high margin of safety. In the same study, there were no cases of pressure increases that necessitated the discontinuation of steroid therapy.[1] Another post-cataract extraction study reported higher pressures with dexamethasone than with FML.[2]

Patient comfort is another reason to choose FML. The Liquifilm® vehicle soothes and lubricates ocular tissues and doesn't interfere with epithelial regeneration.

FML® (fluorometholone) **works well under pressure.**

INDICATIONS—For steroid responsive inflammation of the palpebral and bulbar conjunctiva, cornea and anterior segment of the globe. CONTRAINDICATIONS—Acute superficial herpes simplex keratitis. Fungal diseases of ocular structures. Vaccinia, varicella and most other viral diseases of the cornea and conjunctiva. Tuberculosis of the eye. Hypersensitivity to the constituents of this medication. WARNINGS—Steroid medication in the treatment of herpes simplex keratitis (involving the stroma) requires great caution; frequent slit-lamp microscopy is mandatory. Prolonged use may result in glaucoma, damage to the optic nerve, defects in visual acuity and fields of vision, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from fungi or viruses liberated from ocular tissue. In those diseases causing thinning of the cornea or sclera, perforation has been known to occur with use of topical steroids. Acute purulent untreated infection of the eye may be masked or activity enhanced by presence of steroid medication. Safety and effectiveness have not been demonstrated in children of the age group 2 years or below. *Use in Pregnancy:* Safety of the use of topical steroids during pregnancy has not been established. PRECAUTIONS—As fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid applications, fungus invasion must be suspected in any persistent corneal ulceration where a steroid has been used or is in use. Intraocular pressure should be checked frequently. ADVERSE REACTIONS—Glaucoma with optic nerve damage, visual acuity or field defects, posterior subcapsular cataract formation, secondary ocular infection from pathogens liberated from ocular tissues, perforation of the globe.

REFERENCES:

1. Castroviejo, Ramon. The control of postoperative inflammation with fluorometholone. Klin Mbl Augenheilk 166:518-520, 1975.
2. Marun, Edgard. Evaluation of a new topical steroid. Fluorometholone, in the postoperative course of the cataract operation. Ann Ophthalmol 7:139-142, 1975.

ALLERGAN Pharmaceuticals, Inc., Irvine, California 92713, U. S. A.

40506

UNCOMPLICATED
ORDERING.

II-2.5
AOSOFT®
(tetrafilcon
Warning: If any eye abrasion, ulceration, irritation or infection is present, a physician should be consulted immediately.

Ordering a replacement AOSOFT® (tetrafilcon A) Hydrophilic Contact Lens is easy. Your AOSOFT distributor maintains all patient warranties, so a simple telephone call is all that's required. With the AOSOFT lens there's less paperwork for you to handle, so you have more time left to devote to your patients.

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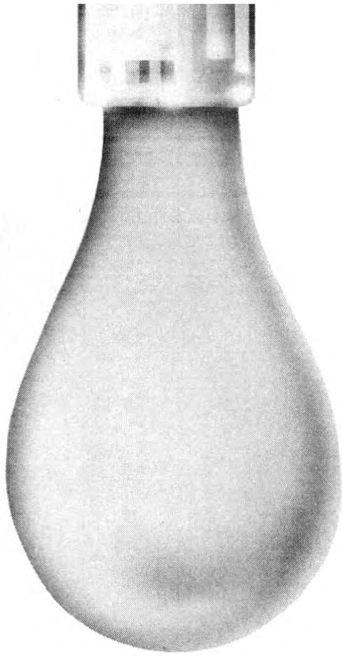
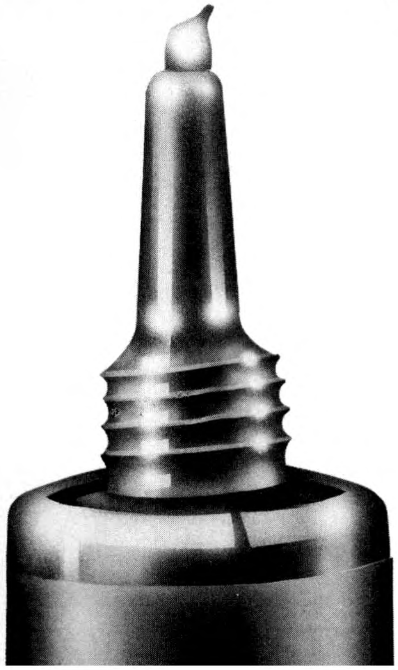


Cortisporin® Ophthalmic Suspension
(Sterile)

For your eyes only!

Cortisporin® Ophthalmic Ointment
(Sterile)



☐ ***Because of your specialized knowledge and training,*** we believe that only you should prescribe the Cortisporin® ophthalmic products—and that's why they are promoted only to ophthalmologists.

☐ ***For your patients*** the Cortisporin ophthalmic products provide an effective therapeutic spectrum. The antibacterials act together against susceptible bacterial pathogens commonly involved in the ophthalmic infections accessible to topical therapy.* The hydrocortisone component gently and rapidly relieves signs and symptoms of inflammation and inhibits corneal neovascularization.

☐ ***Economical, too,*** with the two sterile forms providing convenient day and night coverage at lower cost than most other major brands of similar activity.

Cortisporin® Ophthalmic Suspension Sterile
(Polymyxin B–Neomycin–Hydrocortisone)

Each cc contains Aerosporin® (Polymyxin B Sulfate) 10,000 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base), hydrocortisone 10 mg (1%), thimerosal (preservative) 0.001%. The vehicle contains the inactive ingredients cetyl alcohol, glyceryl monostearate, liquid petrolatum, polyoxyl 40 stearate, propylene glycol and purified water. Bottle of 5 cc with sterile dropper.

Cortisporin® Ophthalmic Ointment Sterile
(Polymyxin B–Bacitracin–Neomycin–Hydrocortisone)

Each gram contains Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base), hydrocortisone 10 mg (1%), special white petrolatum qs. Tube of 1/8 oz with ophthalmic tip.

***INDICATIONS:** Based on a review of these drugs by the National Academy of Sciences–National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: For the treatment of nonpurulent bacterial infections of the eye due to organisms sensitive to the antibiotic ingredients of the drugs and when the anti-inflammatory action of the hydrocortisone is indicated as in nonpurulent bacterial, allergic, vernal and phlyctenular conjunctivitis; nonpurulent blepharitis; interstitial, sclerosing, postoperative or acne rosacea keratitis; superficial chemical and thermal burns of the cornea.

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: These products are contraindicated in acute purulent conjunctivitis and blepharitis; tuberculous, fungal or viral lesions of the skin or eye, including herpes simplex, dendritic keratitis, vaccinia or varicella; and conditions involving the posterior segment of the eye. They are also contraindicated in those individuals who have shown hypersensitivity to any of their components.

PRECAUTIONS: Extended ophthalmic use of topical steroid therapy may result in glaucoma with possible damage of the optic nerves, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from fungi or viruses liberated from ocular tissues. It is advisable that intraocular pressure be checked frequently. In those diseases causing thinning of the cornea, perforation has been known to occur with the use of topical steroids. As with any antibiotic preparation, prolonged use may result in the overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Articles in the current medical literature indicate an increase in the prevalence of persons allergic to neomycin. The possibility of such a reaction should be borne in mind.

Complete literature available on request from Professional Services Dept. PML.

Wellcome

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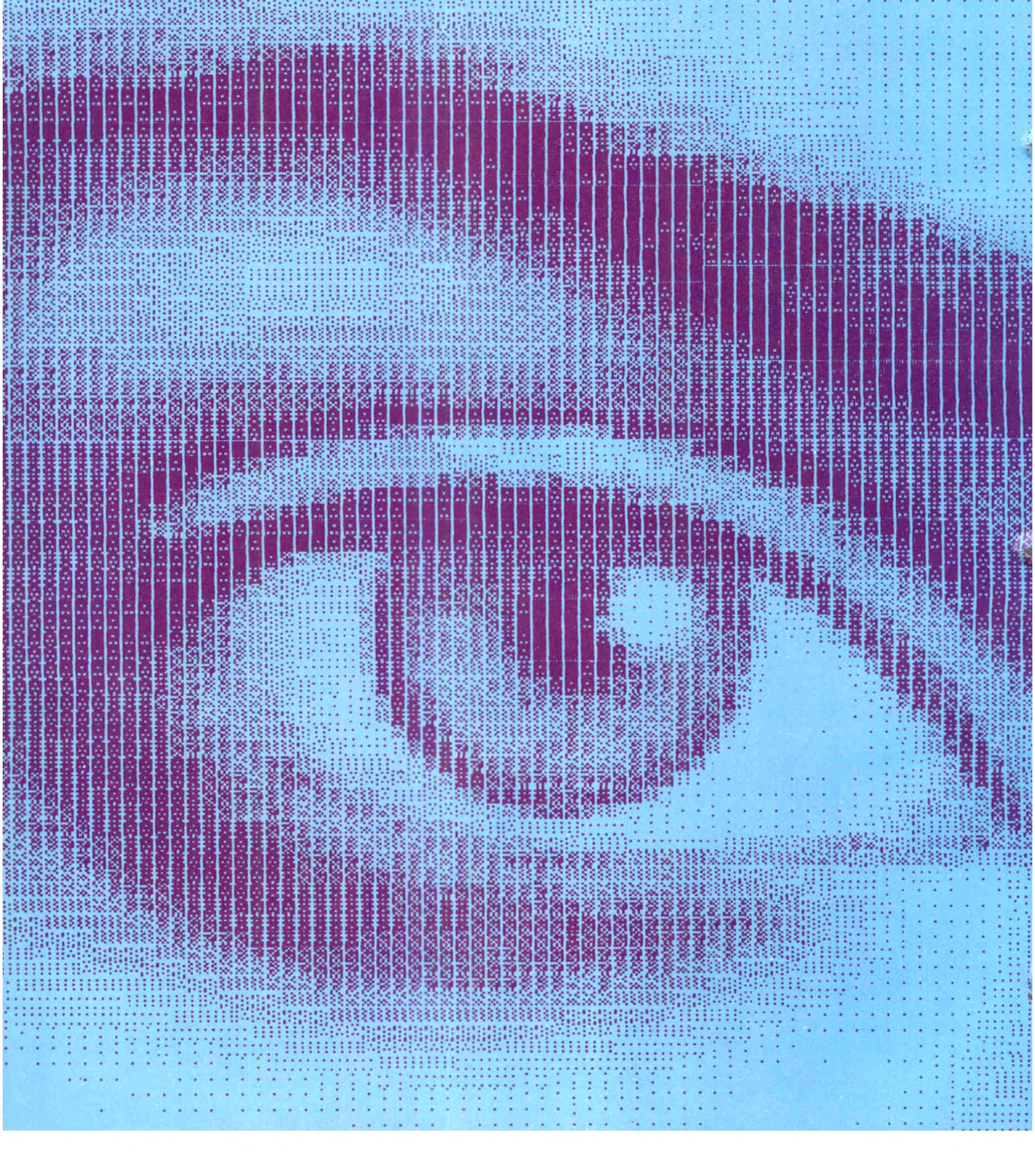
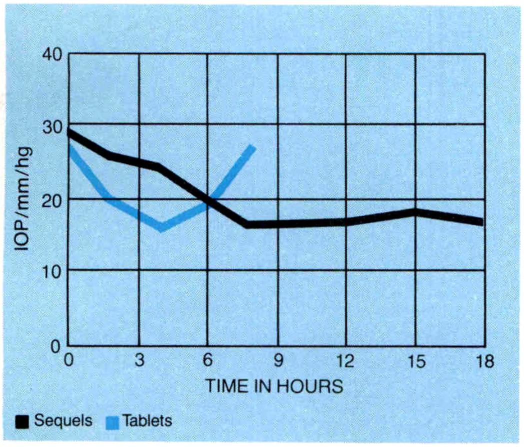
North Carolina 27709

THROUGH THE EYE OF THE COMPUTER

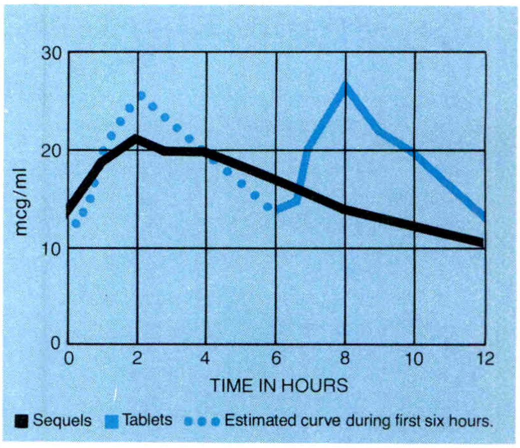
The calculated role of bioavailability in I.O.P. control



A Computer Report



Effect on I.O.P. over an 18-hour period after a single oral dose of DIAMOX SEQUELS, based on computer analysis of patient I.O.P. records.



Steady-state blood levels of acetazolamide over a period of 12 hours following one DIAMOX SEQUELS Capsule twice a day, compared with administration of 250 mg tablets *q.i.d.* Based on computer analysis of patient blood samples.

DIAMOX® SEQUELS®

ACETAZOLAMIDE

Sustained Release Capsules 500 mg.

Proven bioavailability for effective control of I.O.P. with convenient *b.i.d.* dosage

Elaborate and complex manufacturing processes achieve the correct blend of coated and uncoated granules for sustained and optimal release of DIAMOX SEQUELS medication. Equally important, computer analysis confirms batch to batch bioavailability after manufacture. DIAMOX SEQUELS—convenient *b.i.d.* dosage that produces a predictable response in your patients.

Indications: ***Tablets and Parenteral only:*** For adjunctive treatment of: edema due to congestive heart failure; drug-induced edema; centrencephalic epilepsies (petit mal, unlocalized seizures). *All forms:* Chronic simple (open angle) glaucoma, secondary glaucoma, and preoperatively in acute angle closure glaucoma where delay of surgery is desired in order to lower intraocular pressure.

Contraindications: When sodium and/ or potassium serum levels are depressed, in marked kidney and liver disease or dysfunction, suprarenal gland failure and hyperchloremic acidosis. Long-term use in chronic noncongestive angle closure glaucoma.

Warning: Although teratogenic and embryocidal effects demonstrated in mice at more than ten times the equivalent therapeutic doses have not been evidenced in humans, do not use DIAMOX in pregnancy, especially during the first trimester, unless expected benefits outweigh these potential adverse effects.

Precautions: Increasing the dose may increase drowsiness and paresthesia and decrease diuresis. Adverse reactions common to all sulfonamide derivatives may occur: fever, rash, crystalluria, renal calculus, bone marrow depression, thrombocytopenic purpura, hemolytic anemia, leukopenia, pancytopenia, agranulocytosis. Early detection is advised and if such occur, discontinue drug and institute appropriate therapy.

Adverse Reactions: Short-term therapy; (minimal) paresthesias, particularly a "tingling" feeling in the extremities; some loss of appetite, polyuria, drowsiness, confusion. Long-term therapy: An acidotic state may supervene usually corrected by bicarbonate. Transient myopia. *Other:* (occasional) urticaria, melena, hematuria, glycosuria, hepatic insufficiency, flaccid paralysis, convulsions.

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A Division of American Cyanamid Company
Pearl River, New York 10965

019-9

Ophthalmologist

The Ministry of Health of the State of Bahrain would like applications for the above post in the new Salmaniya Medical Center. Applicants should possess a speciality in retinal diseases and surgery. Terms and conditions of employment include:

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3. Settling-in and car purchase allowances
4. Generous education allowances
5. Liberal leave policy/mid-term travel
6. Year-end gratuity/two year contract
7. State-furnished medical care
8. Paid family air fares and air cargo entitlement.

Applicants will be notified if they are selected for interview. All applications should include a passport size photograph and copies of appropriate certificates.

Address and airmail to:

S.H. Clarke,
Personnel Advisor,
Civil Service Bureau,
P.O. Box 1066, Manama,
Bahrain, Arabian Gulf.

FACULTY POSITION AVAILABLE

General Ophthalmologist with subspecialty training in retina is sought for well established University Department of Ophthalmology, academic rank commensurate with credentials. Qualifications include successful completion of approved eye residency program plus fellowship experience in retina. Duties include teaching, patient care and research. Salary based on strict full time rank. Send curriculum vitae, bibliography and references to John L. Keltner, M.D., Department of Ophthalmology, University of California, Davis, CA 95616. Open until filled.

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AFFIRMATIVE ACTION EMPLOYER

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brand of
gentamicin sulfate, U.S.P.

OPHTHALMIC

Solution-Sterile
Ointment-Sterile

Each ml. or gram contains gentamicin sulfate equivalent to 3.0 mg. gentamicin.

DESCRIPTION Gentamicin sulfate is a water-soluble antibiotic of the aminoglycoside group active against a wide variety of pathogenic gram-negative and gram-positive bacteria.

GARAMYCIN **Ophthalmic Solution** is a sterile, aqueous solution buffered to approximately pH 7 for use in the eye. Each ml. contains gentamicin sulfate (equivalent to 3.0 mg. gentamicin), disodium phosphate, monosodium phosphate, sodium chloride, and benzalkonium chloride as a preservative.

GARAMYCIN **Ophthalmic Ointment** is a sterile ointment, each gram containing gentamicin sulfate (equivalent to 3.0 mg. gentamicin) in a bland base of white petrolatum, with methylparaben and propylparaben as preservatives.

ACTIONS The gram-positive bacteria against which gentamicin sulfate is active include coagulase-positive and coagulase-negative staphylococci, including certain strains that are resistant to penicillin; Group A beta-hemolytic and nonhemolytic streptococci; and *Diplococcus pneumoniae*. The gram-negative bacteria against which gentamicin sulfate is active include certain strains of *Pseudomonas aeruginosa*, indole-positive and indole-negative *Proteus* species, *Escherichia coli*. *Klebsiella pneumoniae* (Friedlander's bacillus), *Haemophilus influenzae* and *Haemophilus aegyptius* (Koch-Weeks bacillus), *Aerobacter aerogenes*. *Moraxella lacunata* (diplobacillus of Morax-Axenfeld), and *Neisseria* species, including *Neisseria gonorrhoeae*. Although significant resistant organisms have not been isolated from patients treated with gentamicin at the present time, this may occur in the future as resistance has been produced with difficulty *in vitro* by repeated exposures.

INDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are indicated in the topical treatment of infections of the external eye and its adnexa caused by susceptible bacteria. Such infections embrace conjunctivitis, keratitis and keratoconjunctivitis, corneal ulcers, blepharitis and blepharoconjunctivitis, acute meibomianitis, and dacryocystitis.

CONTRAINDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are contraindicated in patients with known hypersensitivity to any of the components.

WARNINGS GARAMYCIN **Ophthalmic Solution** is not for injection. It should never be injected subconjunctivally, nor should it be directly introduced into the anterior chamber of the eye.

PRECAUTIONS Prolonged use of topical antibiotics may give rise to overgrowth of nonsusceptible organisms, such as fungi. Should this occur, or if irritation or hypersensitivity to any component of the drug develops, discontinue use of the preparation and institute appropriate therapy.

Ophthalmic ointments may retard corneal healing.

ADVERSE REACTIONS Transient irritation has been reported with the use of GARAMYCIN **Ophthalmic Solution.**

Occasional burning or stinging may occur with the use of GARAMYCIN **Ophthalmic Ointment.**

DOSAGE AND ADMINISTRATION GARAMYCIN **Ophthalmic Solution:** instill one or two drops into the affected eye every four hours. In severe infections, dosage may be increased to as much as two drops once every hour.

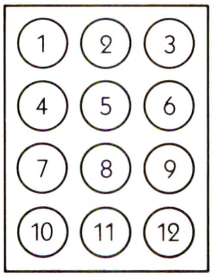
GARAMYCIN **Ophthalmic Ointment:** apply a small amount to the affected eye two to three times a day.

HOW SUPPLIED GARAMYCIN **Ophthalmic Solution—Sterile,** 5-ml. plastic dropper bottle, sterile, boxes of one and six. Store away from heat.

GARAMYCIN **Ophthalmic Ointment—Sterile,** ⅛-ounce tube, boxes of one and six. Store away from heat.

NOVEMBER 1973 10082900

For complete details, consult package insert or literature available from your Schering Representative; or Professional Services Department, Schering Corporation, Kenilworth, New Jersey 07033.



1. Staphylococcus sp.
2. Streptococcus sp.
3. H. influenzae
4. H. aegyptius
5. M. lacunata
6. Neisseria sp.
7. Proteus sp.
8. P. aeruginosa (certain strains)
9. E. coli
10. A. aerogenes
11. K. pneumoniae
12. D. pneumoniae

Schering Corporation
Kenilworth, N.J. 07033

SWW-1320 R1

Solution-Sterile/Ointment-Sterile
Each ml. or gram contains gentamicin sulfate equivalent to 3.0 mg. gentamicin.

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GARAMYCIN® OPHTHALMIC

brand of
gentamicin sulfate, U.S.P.

STANDS THE TEST OF TIME THROUGHOUT A BROAD RANGE OF OCULAR INFECTIONS*

*Due to susceptible organisms. In vitro data is not necessarily indicative of clinical effectiveness. Sensitivity data may vary among institutions and may depend on the technique used in sensitivity testing.

Please see adjacent product information.

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Timoptic®

(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION

the only agent you'll need for most patients with chronic open-angle glaucoma.

...alone

TIMOPTIC alone:
greater efficacy and safety

- Significantly more effective than pilocarpine or epinephrine in reducing IOP.*
- Adverse effects commonly caused by pilocarpine and epinephrine (as reported in the literature) rarely encountered with TIMOPTIC.

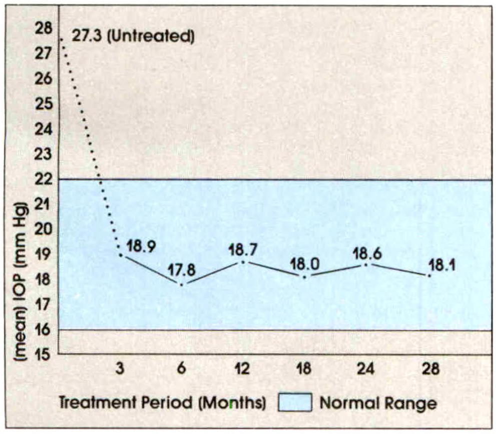
TIMOPTIC (Timolol Maleate, MSD) alone controlled 76% of patients (485 of 638)

Based on clinical studies of 638 patients maintained on therapy from 14 to 477 days:

- approximately 76% (485 of 638) of patients satisfactorily managed on TIMOPTIC alone (IOP less than 22 mm Hg).
- approximately 93% (593 of 638) of patients satisfactorily managed with TIMOPTIC alone or administered concomitantly with other antiglaucoma agents.

TIMOPTIC alone:
highly effective long-term therapy

TIMOPTIC alone maintained control of IOP with no evidence of tolerance observed in 102 patients for over 2 years



In a long-term maintenance study, the mean IOP was reduced from an untreated level of 27.3 mm Hg to 18.9 mm Hg after three months of treatment. The mean IOP remained below this level at all subsequent intervals up to 28 months.

TIMOPTIC alone:
greater patient comfort and convenience

TIMOPTIC alone offers:

- little or no change in pupil size
- little or no blurring of vision
- little or no effect on tear production
- little or no hyperemia
- no dimming of vision
- no night blindness
- simple "one-drop" b.i.d. dosage may encourage long-term compliance

There are significantly fewer and less severe side effects than with pilocarpine or epinephrine.*

TIMOPTIC
compatible with other agents:

Helpful in the management of a broader range of patients.

- For the patient who needs reduction of IOP greater than that provided by one drop 0.5% TIMOPTIC twice a day, concomitant therapy with pilocarpine and other miotics, and/or epinephrine, and/or systemically administered carbonic anhydrase inhibitors, such as acetazolamide, can be used with TIMOPTIC.
- In some patients who respond inadequately to maximum available drug therapy, the addition of TIMOPTIC may result in satisfactory management of intraocular pressure, thus possibly postponing the need for surgery.

TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product. Occasionally, mild ocular irritation has been reported.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma. TIMOPTIC is not recommended for use in children at this time.

---

*Based on controlled multiclinic studies, evaluating over 1000 patients.

MSD
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For a brief summary of Prescribing Information, please see following page.

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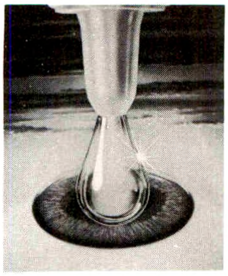

Timoptic®

(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION

Rx
Timoptic
(Timolol
Maleate | MSD)
0.25%
one drop
in each eye
b.i.d.

a beta-adrenergic blocking agent in ophthalmic solution

a significant advance in the treatment of:

- chronic open-angle glaucoma
- ocular hypertension
- aphakic glaucoma
- some patients with secondary glaucoma
- some patients who respond inadequately to multiple antiglaucoma drug therapy

How to start patients on TIMOPTIC:
Usual starting dosage: one drop 0.25% TIMOPTIC in each eye twice a day.

How to transfer from current therapy to TIMOPTIC:
1. On the first day: continue with the agent(s) already being used and add one drop 0.25% TIMOPTIC in each eye b.i.d.

2. On the second day: discontinue the previously used agent(s) and continue with TIMOPTIC in each eye b.i.d.

If clinical response is not adequate:
Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC twice a day in each eye. If the intraocular pressure is maintained at satisfactory levels, the dosage schedules may be changed to one drop once a day in each eye. Dosages above one drop of 0.5% TIMOPTIC twice a day generally have not been shown to produce further reduction of IOP.

Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.

CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.
PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchial asthma; sinus bradycardia and greater than first-degree block; cardiogenic shock; right ventricular failure secondary to pulmonary hypertension; congestive heart failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.
Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.
Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.
ADVERSE REACTIONS: Occasionally, signs and symptoms of mild ocular irritation have been reported. Slight reduction of the resting heart rate (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed in some patients. Local hypersensitivity reactions have occurred rarely.
Note: TIMOPTIC has been used in patients with glaucoma wearing conventional (PMMA) hard contact lenses, and has generally been well tolerated. TIMOPTIC has not been studied in patients wearing lenses made with materials other than PMMA.
HOW SUPPLIED:
TIMOPTIC Ophthalmic Solution, 0.25%
TIMOPTIC Ophthalmic Solution, 0.5%
Both are available in 5-ml plastic OCUMETER® Ophthalmic Dispenser with a controlled drop tip.
J8TMO5 (DC7046004)

MSD
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For more detailed information, consult your MSD Representative or see full Prescribing Information.
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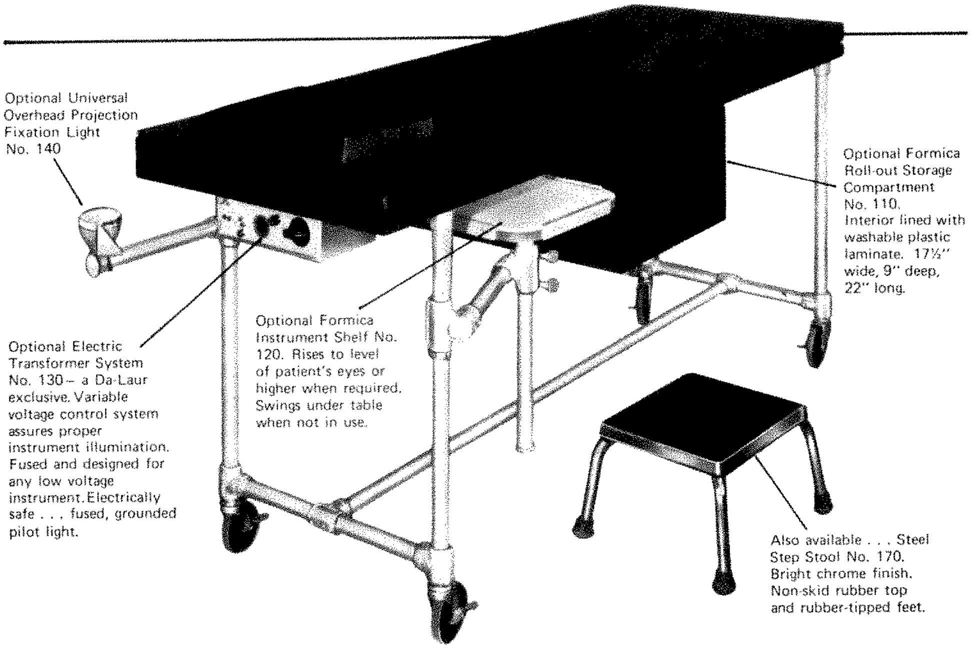
Optional Universal Overhead Projection Fixation Light No. 140
Optional Formica Roll-out Storage Compartment No. 110. Interior lined with washable plastic laminate. 17½" wide, 9" deep, 22" long.
Optional Formica Instrument Shelf No. 120. Rises to level of patient's eyes or higher when required. Swings under table when not in use.
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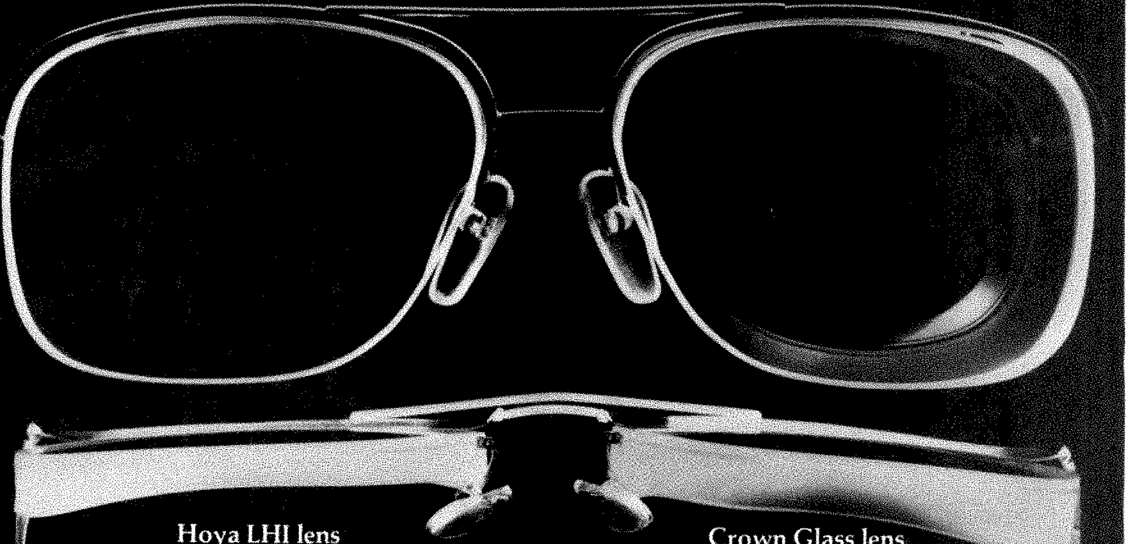
The Hoya LHI lens.
The difference is clear.



Hoya LHI lens
1.702 index
−8.00D, 2.2mmCT

Crown Glass lens
1.523 index
−8.00D, 2.2mmCT

Chances are the high-prescription glass lenses you've been prescribing improve your patient's eyesight, but are thick and unattractive. That's why Hoya invented the thin LHI lens, (1.702 index). Now people with high plus or minus prescriptions (+3 to +10D and −4 to −20D ranges) can feel comfortable about wearing glasses.

LHI lenses are not only thinner and lighter than traditional glass lenses, they are vacuum-coated front and back with Hoya 99 Multicoated.* This combination reduces unsightly myopic rings and transmits over 99% of usable visible light. In addition, Hoya's LHI lens has a color dispersion factor of 40.2—the lowest dispersion of any comparable lens—which greatly reduces color aberration for significant improvement in visual acuity. These combinations make Hoya the leader in the high index lens field.

The lenses are finished or semi-finished, come only in glass, and are single vision. (Sizes 60 and 65 mm).

The LHI lenses with Hoya 99 Multicoated. It'll make you look good, too.

HOYA LENS

*For further Hoya 99 Multicoated and LHI lens information call or write: Hoya Lens of America, Inc. 970 Knox Street, Torrance, CA 90502/National Wats: 800-421-1993/California Wats: 800-262-1587

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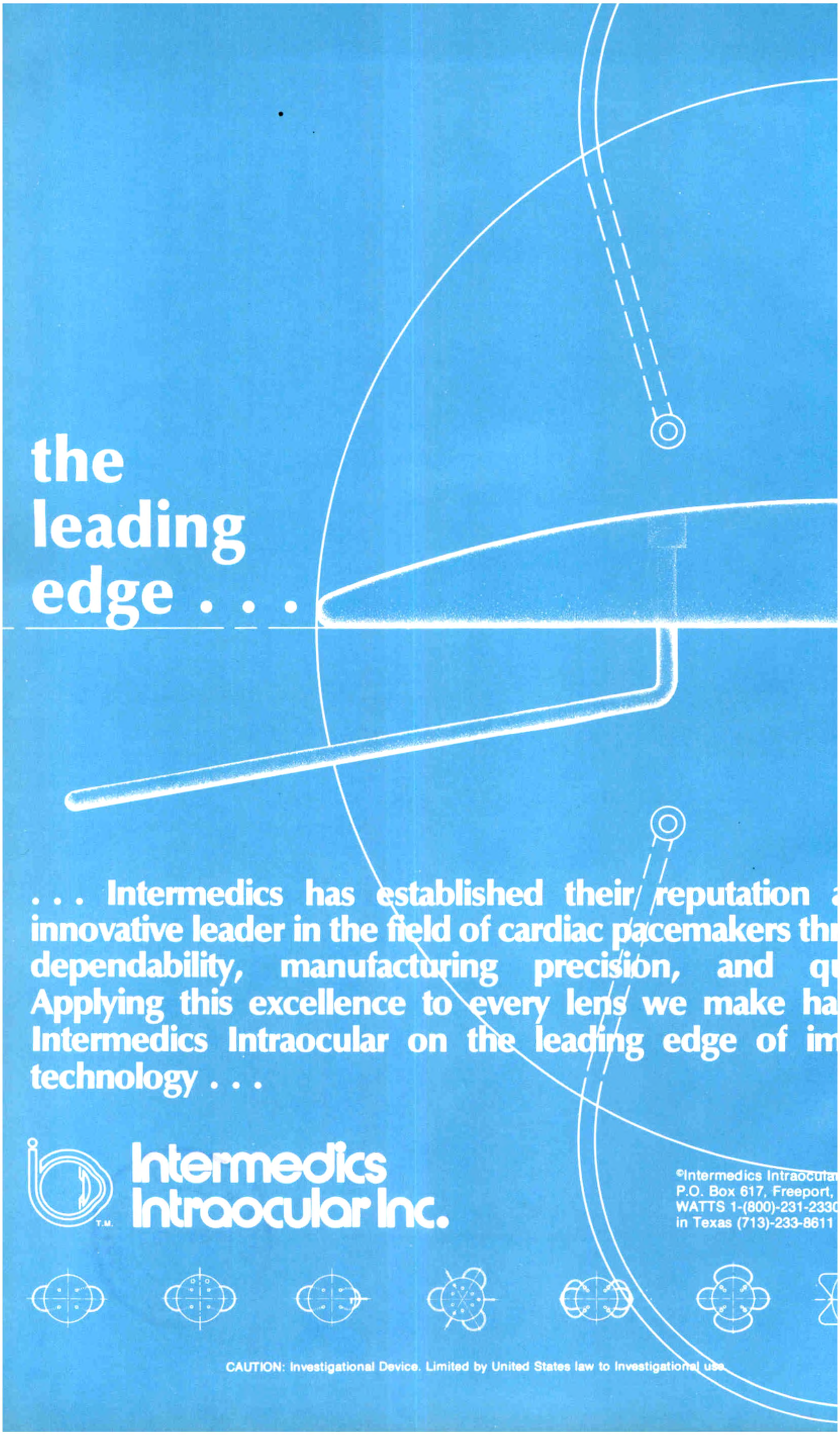

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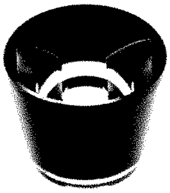
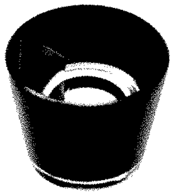
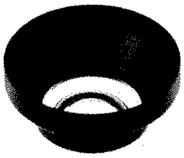
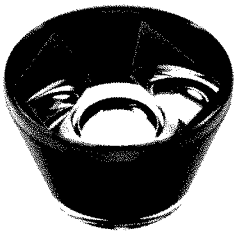
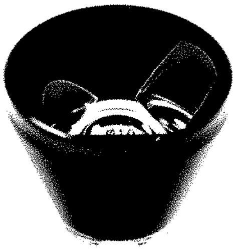

One-mirror gonio lens

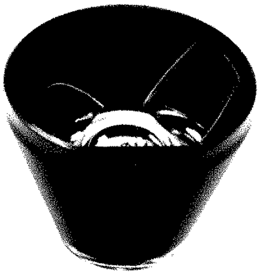

Two-mirror gonio lens


Macula lens


Small Three-mirror lens

Large Three-mirror lens


Three-mirror lens
for babies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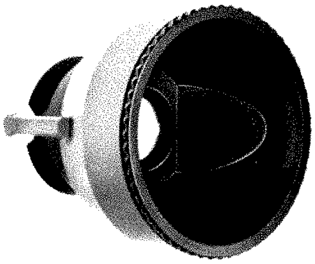
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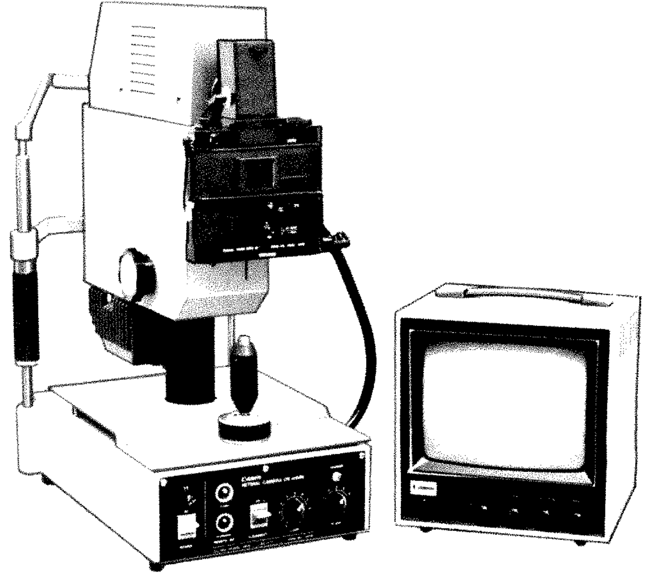

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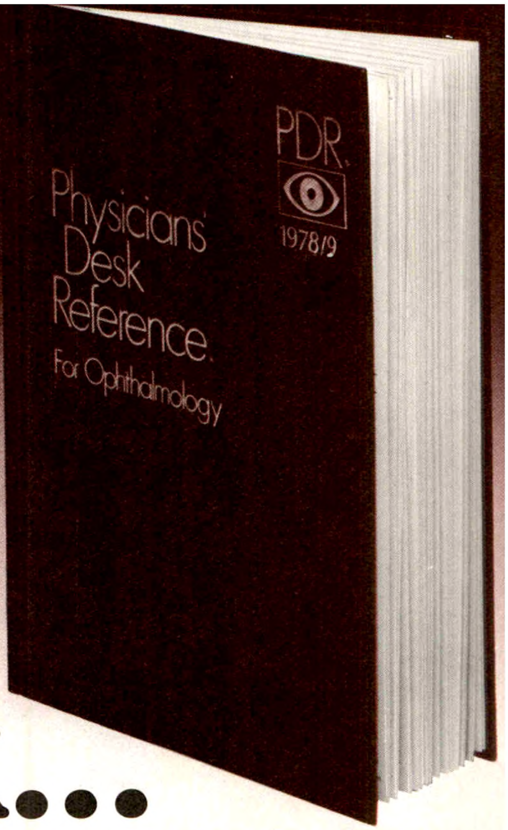

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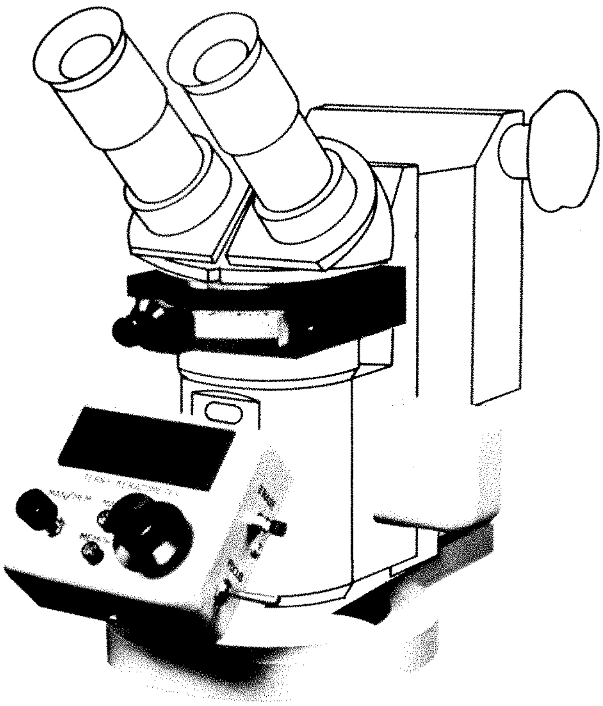

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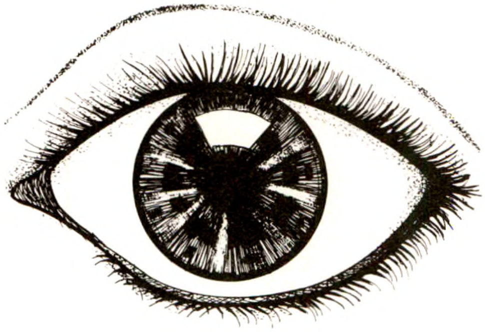

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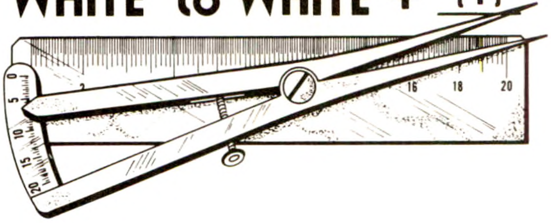

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CONTRAINDICATIONS Hypersensitivity to sulfonamide preparations contraindicates the use of Sodium SULAMYD
PRECAUTIONS The solutions are incompatible with silver preparations Ophthalmic ointments may retard corneal healing Non-susceptible organisms including fungi may proliferate with the use of these preparations Sulfonamides are inactivated by the para-aminobenzoic acid present in purulent exudates Sulfonamide sensitivity reactions may occur
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brand of prednisolone acetate U S P and sulfacetamide sodium U S P
Ophthalmic Suspension and Ointment Sterile Each ml or Gm contains 5 mg prednisolone acetate and 100 mg sulfacetamide sodium

INDICATIONS *Suspension* Based on a review of this drug by the National Academy of Sciences - National Research Council and or other information FDA has classified the indications as follows
"Possibly" effective for the treatment of the following inflammatory and allergic conditions affecting the eyelids and anterior segment of the eye EYELIDS allergic blepharitis blepharitis associated with seborrheic dermatitis and other nonpurulent forms of conjunctivitis including those associated with hay fever and conjunctivitis due to physical agents such as foreign bodies chemicals acids alkalies and other irritants CORNEA SCLERA IRIS AND UVEA interstitial postoperative and sclerosing keratitis chemical and thermal burns of the cornea corneal ulcer herpes zoster ophthalmicus phlyctenular keratoconjunctivitis corneal neovascularization scleritis episcleritis acute chronic and traumatic iridocyclitis Final classification of the less-than-effective indications requires further investigation

Ointment METIMYD is indicated in the following inflammatory and allergic conditions affecting the eyelids and anterior segment of the eye
Eyelids allergic blepharitis blepharitis associated with seborrheic dermatitis other nonpurulent types of blepharitis
Conjunctiva allergic conjunctivitis ie vernal phlyctenular and other nonpurulent forms of conjunctivitis including those associated with hay fever conjunctivitis due to physical agents such as foreign bodies chemicals acids alkalies and other irritants
Cornea Sclera Iris Uvea interstitial postoperative and sclerosing keratitis chemical and thermal burns of the cornea corneal ulcer herpes zoster ophthalmicus phlyctenular keratoconjunctivitis corneal neovascularization scleritis episcleritis acute chronic and traumatic iridocyclitis
In deep-seated infections such as endophthalmitis panophthalmitis and orbital cellulitis or when systemic infection threatens specific oral antibiotic sulfonamide therapy should be employed Local treatment may be used as adjunctive therapy
CONTRAINDICATIONS The contraindications for METIMYD are the same as those for other corticosteroid sulfonamide preparations Topical ophthalmic corticosteroid preparations and combinations are contraindicated in early acute herpes simplex and the early acute stages of most other viral diseases of the cornea and conjuctiva active tuberculosis of the anterior segment of the eye fungal disease of the cornea conjunctiva and lids acute purulent untreated infections of the eye which like other diseases caused by microorganisms may be masked or enhanced by the presence of the steroid individuals with known sensitivity to any of the ingredients
PRECAUTIONS Extended use of topical steroid therapy may cause increased intraocular pressure in certain individuals In prolonged therapy it is advisable that intraocular pressure be checked frequently In those diseases causing thinning of the cornea perforation has been known to have occurred with the use of topical steroids As with any antibacterial preparation prolonged use may result in overgrowth of non-susceptible organisms including fungi If superinfection occurs appropriate measures should be instituted Sensitivity reactions may occur in certain individuals If signs of sensitivity develop discontinue use
The protracted use of topical corticosteroids in the eye reportedly has been rarely associated with the development of posterior subcapsular cataracts
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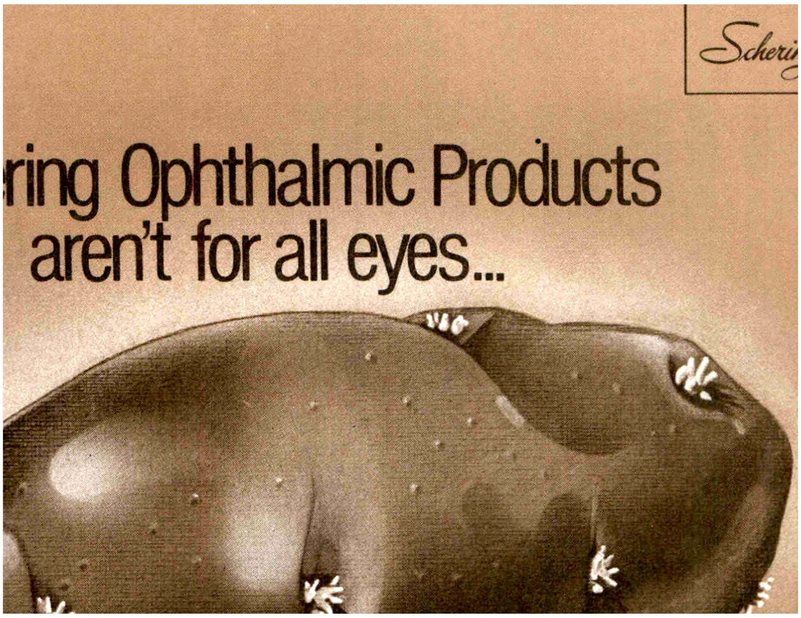
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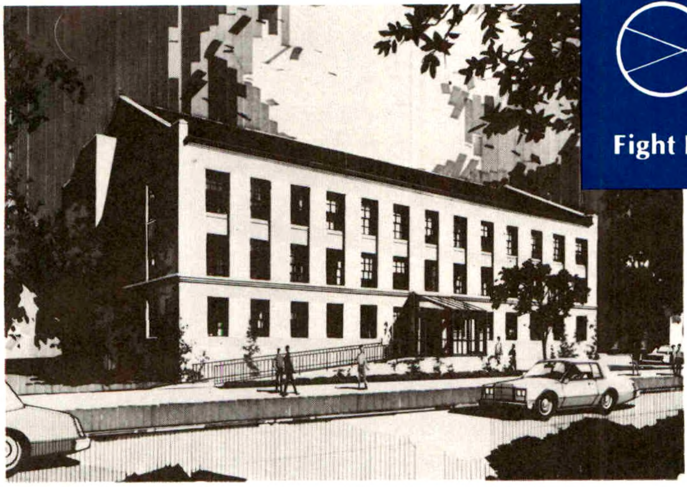

Department of Ophthalmology
Pritzker School of Medicine
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September 14 and 15, 1979
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Ophthalmology 1979 and the Decade to Come

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Samuel D. McPherson, M.D.
David Paton, M.D.
David Shoch, M.D.

Faculty

Karl J. Fritz, Ph.D., M.D.
Royce O. Johnson, M.D.
Harvey Z. Klein, M.D.
Peter H. Morse, M.D.
Frank W. Newell, M.D.
Joel M. Pokorny, Ph.D.
Vivianne C. Smith, Ph.D.
Brenda J. Tripathi, Ph.D.
Ramesh C. Tripathi, M.D., Ph.D.

Friday, September 14

Program — Drake Hotel, 100 E. Lake Shore Drive	8:30-5:00
Reception — Eye Research Laboratories, 939 E. 57th Street	5:00-7:00
Banquet — Quadrangle Club, 1155 E. 57th Street	7:00

Saturday, September 15

Program — Drake Hotel, 100 E. Lake Shore Drive	8:30-1:00

Registration Fee *(Includes luncheon, banquet, and transportation to and from Eye Research Laboratories):*
$150.00 — Practicing physicians
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12 Category I AMA credits

For Information

Accommodations:
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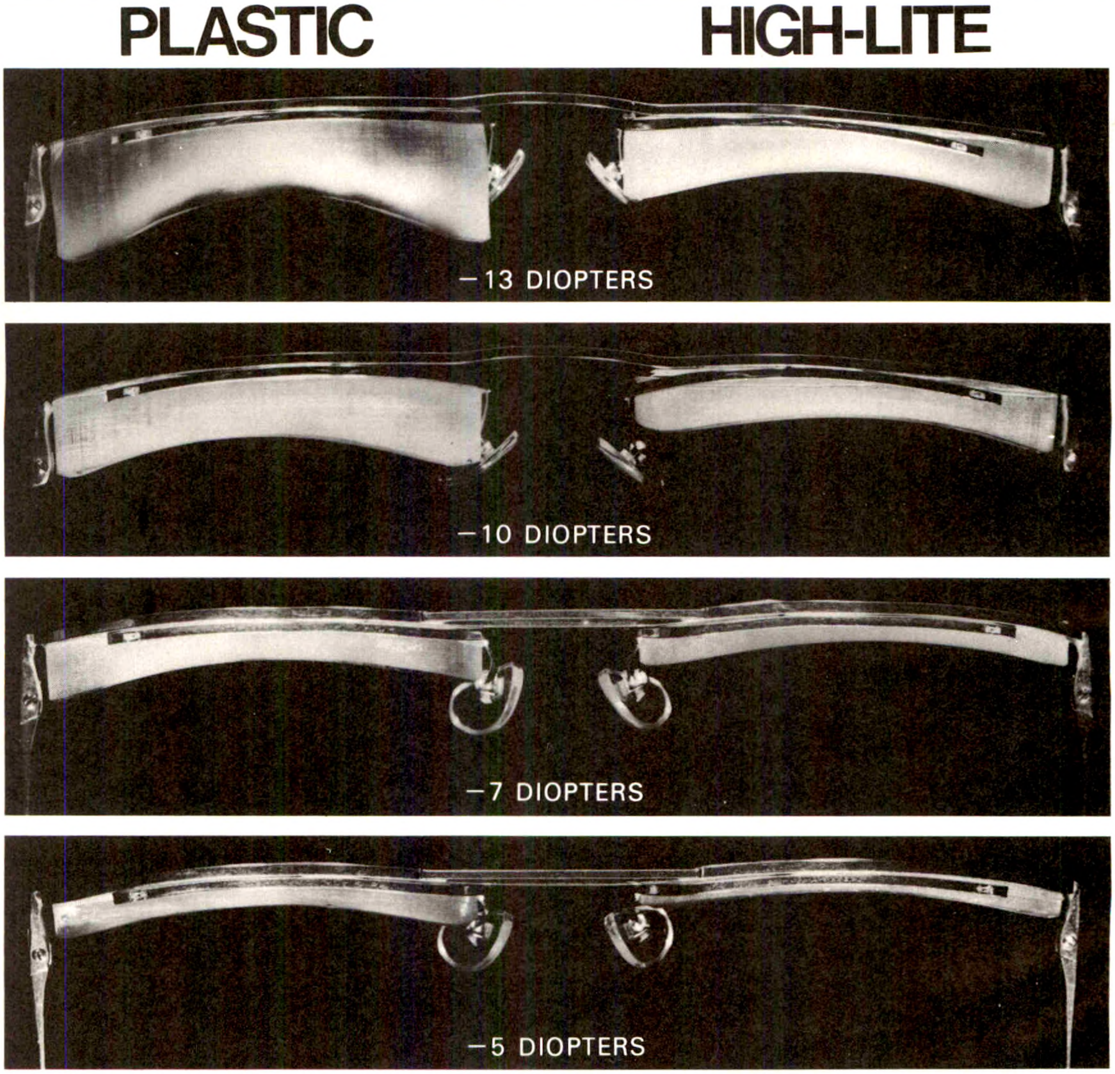

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Bausch & Lomb SOFLENS® (polymacon) **Contact Lenses**

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See full prescribing information which follows on last page.

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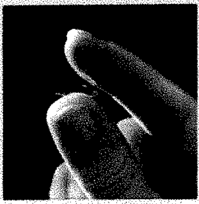

BAUSCH & LOMB SOFLENS® (polymacon) Contact Lenses

VISION CORRECTION USE

DESCRIPTION: The SOFLENS* (polymacon) Contact Lens is a hemispherical flexible shell which covers the cornea and may cover a portion of the adjacent sclera. It consists of 61.4% poly (2-hydroxy-ethyl methacrylate) and 38.6% water by weight when immersed in normal saline. The material has a refractive index of 1.43 and the lens has a visible light transmittance of more than 97%.

ACTIONS: In its hydrated state a SOFLENS®(polymacon) Contact Lens is soft and pliable. When placed on the human cornea, the hydrated SOFLENS®(polymacon)Contact Lens acts as a refracting medium to compensate spherical ametropias.

INDICATIONS: SOFLENS* (polymacon) Contact Lenses are indicated for vision correction use in persons with non-diseased eyes and in aphakia.

CONTRAINDICATIONS: SOFLENS* (polymacon) Contact Lenses are contraindicated in the presence of any active untreated infection or abnormality of the anterior segment of the eye with the exception of ametropia and aphakia.

WARNINGS: Abrasions and Infections—If a lens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present, or any abnormal eye condition is observed concurrent with lens wear, the lens should be removed immediately and a physician consulted.

Infectious corneal ulcers have been reported, usually associated with failure to follow the recommended procedures for care of the lenses.

Aphakic Patients—Aphakic patients should not be fitted with SOFLENS®(polymacon) Contact Lenses during the postoperative period until, in the opinion of the surgeon, the eye has healed completely.

Lens Sanitation and Handling—Persons who require only vision correction and who would not, or could not, adhere to the recommended daily sanitary care of SOFLENS®(polymacon) Contact Lenses or who are unable to place and remove the lenses should not be provided with them. Failure to follow handling and sanitation instructions could lead to serious eye infections which might result in corneal ulcers.

Malfunction and rusting of the metal interior of the BAUSCH & LOMB™ Disinfecting Unit (ASEPTOR®) as well as discoloration and cracking of the lens case has been reported after varying periods of use. If such occurs, appropriate replacement is indicated to avoid interference with the disinfection procedure. Keep the BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) clean and dry at all times.

Medicants and Eye Drops—When the lenses are used by persons requiring only vision correction no ophthalmic solutions or medicants, including conventional (hard) contact lens solutions and eye drops, except BAUSCH & LOMB™ Lens Lubricant, should be used by SOFLENS* (polymacon) Contact Lens wearers prior to placement, or while the lens is in place, on the eye. Also, no solutions, including conventional (hard) contact lens solutions other than daily cleaner, lens lubricant, saline, and the solution made from the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets are to be used on a SOFLENS* (polymacon) Contact Lens when the lens is off the eye.

Wearing Restrictions—SOFLENS® (polymacon) Contact Lenses when used only for vision correction should be removed before sleeping or swimming and in the presence of noxious and irritating vapors. Insufficient lacrimal secretions or hypoesthesia may be restricting factors to the wearing of soft contact lenses.

PRECAUTIONS: Storage—SOFLENS* (polymacon) Contact Lenses must be stored ONLY in saline solution. If left exposed to air, the lenses will dehydrate, become brittle, and break readily. If a lens dehydrates, it should be resoaked in saline solution until it returns to its soft, pliable state which may take as long as forty minutes.

Fresh saline solution must be used DAILY for cleaning, storing, and disinfection of lenses. The carrying case must be emptied and refilled with fresh saline solution just before disinfecting the lenses.

If using BAUSCH & LOMB™ Salt Tablets to make saline solution, the solution **MUST BE PREPARED FRESH DAILY.** As this saline is not aseptic, it should not be used to clean a lens that is to be replaced immediately on the eye, nor should this solution ever be placed in the eye. If the patient wishes to remove a lens, clean it, or wet it and replace it on the eye, the solution in the carrying case should be used, as it will have been disinfected, or Preserved Saline Solution may be used.

Hygiene—Hands must be washed, rinsed thoroughly, and dried with a lint-free towel before handling the lenses.

Cosmetics, lotions, soaps, and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the hair spray has settled.

Handling—A SOFLENS®(polymacon) Contact Lens may be damaged by nicking or tearing if care is not exercised during placement on or removal from the eye, replacing or removing it from the carrying case or in the cleaning process. Lenses must be placed very carefully in the carrying case to avoid damaging the edges of the lenses.

Disinfecting—Fresh saline solution must be used DAILY for cleaning, storing and disinfection of lenses. If using BAUSCH & LOMB™ Salt Tablets to make saline solution, the solution MUST BE PREPARED FRESH DAILY. After removal from the eye, the SOFLENS* (polymacon) Contact Lens must be rubbed gently with Daily Cleaner then rinsed with saline solution to remove mucus and film from the lens surface. The BAUSCH & LOMB™ Lens Carrying Case (LENSGARD™) must be emptied and refilled with fresh saline solution just before disinfecting the lenses.

The causes and nature of deposits formed on the surfaces of some lenses have not been completely evaluated. However, some coatings are known to be proteinaceous and others may be oily or greasy film from extraneous agents, such as hair spray or other cosmetics, or from the patient's own lacrimal secretions. Many wearers experience little or no difficulty with such deposits. However, occasionally a wearer, who tends to secrete unusually large amounts of mucus in the lacrimal fluid, may experience a build-up of these deposits within a relatively few weeks, despite adequate cleaning measures. If surface accumulations of non-removable materials persist, professional care should be obtained promptly.

Deposits, characterized as medium or heavy, were found on 17.5 percent of a population of lenses worn an average of 11.2 months, including lenses worn as long as thirty months. The occurrence of these deposits appeared to increase with the duration of lens use. These medium or heavy deposits, when they do exist, can be detected by means of a slit lamp biomicroscope examination. Light deposits, unrelated to length of lens use and of no apparent clinical significance, were observed on approximately one-half of the lenses studied.

The BAUSCH & LOMB™ Lens Carrying Case (LENSGARD™) should be washed at least once a week with hot water and then rinsed thorougjly with saline solution. Soap or other cleaners should never be used to clean the carrying case.

In order to remove protein deposits which may form on the lenses, wearers should use the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets according to the directions for use which accompany the tablets. To help remove tear residues and mucus as well as other deposits that tend to accumulate on the lens surface, patients should use Daily Cleaner according to the directions for use which accompany the product.

Disinfecting with a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is necessary to kill microorganisms.

If a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is not available, the lenses may be disinfected by boiling them in their carrying case in a pan of water for ten minutes. When this boiling method of disinfection is used, the lenses can be damaged if the boiling water is allowed to completely evaporate.

The BAUSCH & LOMB™ Lens Carrying Case (LENSGARD™) must **always** be **2/3 full** of fresh saline solution before disinfecting and must **always** be tightly closed to prevent leakage of the saline from the case and subsequent dehydration of the lenses.

Fluorescein—Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with sterile normal saline solution and wait at least one hour before replacing the lenses. Earlier replacement may cause the lenses to absorb residual fluorescein.

ADVERSE REACTIONS: Serious corneal damage may result from wearing a SOFLENS* (polymacon) Contact Lens which has been soaked in a conventional contact lens solution.

Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritation.

A lens adheres very rarely to an eye as a result of sleeping with the lens on, or wearing a hypotonic lens. If a lens adheres for any reason, apply fresh saline and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema, as manifested by symptoms such as rainbows, halos around lights, or foggy vision, may occur in less than 5% of SOFLENS* (polymacon) Contact Lens wearers. If these symptoms occur, the lenses should be removed and professional consultation obtained.

Excessive tearing, unusual eye secretions, and photophobia are not normal. If these symptoms occur, the patient should be examined to determine their cause.

A faint blue haze, believed to be located in Descemet's membrane, has been reported in the Spokane, Washington area. As yet, the cause is unknown and the phenomenon has not been found elsewhere. The wearers report no subjective symptoms and there is no detectable effect on their visual acuity. There have been approximately 29 cases, and in 10 of these patients the blue haze has cleared or is in various stages of regression.

Neovascularization of the cornea has been observed in some aphakic patients fitted with the SOFLENS®(polymacon) Contact Lens, which may require discontinuation of the lens. Medical consultation should be obtained in such an instance.

LENS REPLACEMENT: Various studies have been conducted to determine the frequency of lens replacements and the reasons for those replacements. These studies show that during the first four months of wear, approximately one quarter of the lenses initially dispensed are replaced; the principal reason for these replacements is lens damage. Lenses are more apt to be damaged while new wearers are learning the prescribed handling and care techniques. After the initial four months of wear, the average lens replacement rate has been found to be approximately one lens per wearer per year. Lens loss, damage and surface deposits were the major causes for lens replacements.

DOSAGE AND ADMINISTRATION: Conventional methods of fitting contact lenses do not apply to SOFLENS®(polymacon) Contact Lenses. For a detailed description of the fitting technique, refer to the SOFLENS Fitting Guide, copies of which are available from: SOFLENS Division, Bausch & Lomb Incorporated, Rochester, N.Y. 14602.

When the lenses are used only for vision correction, there may be a tendency for the patient to overwear the lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to these patients.

Day	Wear Time (hours)	Rest Period (hours)	Wear Time (hours)	Rest Period (hours)	Wear Time (hours)
1	3	1	3	1	3
2	3	1	3	1	3
3	4	1	4	1	4
4	4	1	4	1	4
5	6	1	6	1	4
6	6	1	6	1	4
7	8	1	8		
8	8	1	8		
9	8	1	8		
10	10	1	balance of the waking hours*		
11	12	1	balance of the waking hours*		
12	14	1	balance of the waking hours*		

*Lenses used only for vision correction should never be worn 24 hours a day.

When lenses are dispensed for vision correction, the wearer must be supplied with a lens care kit and must fully understand all lens care and handling instructions. As with any contact lens, regular recall visits are necessary to assure corneal health and wearer compliance with instructions.

HOW SUPPLIED: Each lens is supplied sterile in a glass vial containing sterile normal saline solution. The glass vial is marked with the manufacturing lot number of the lens and the dioptric power (black for plus power lenses; red for minus; gold or white for plano).

Caution: Federal Law Prohibits Dispensing Without a Prescription.

A BAUSCH & LOMB™ Care Kit is available for lens disinfecting, cleaning, and storage.

Complete information on lenses and accessory products can be found in the current SOFLENS Fitting Guide, price list, or sales policy.

SEPTEMBER 1978

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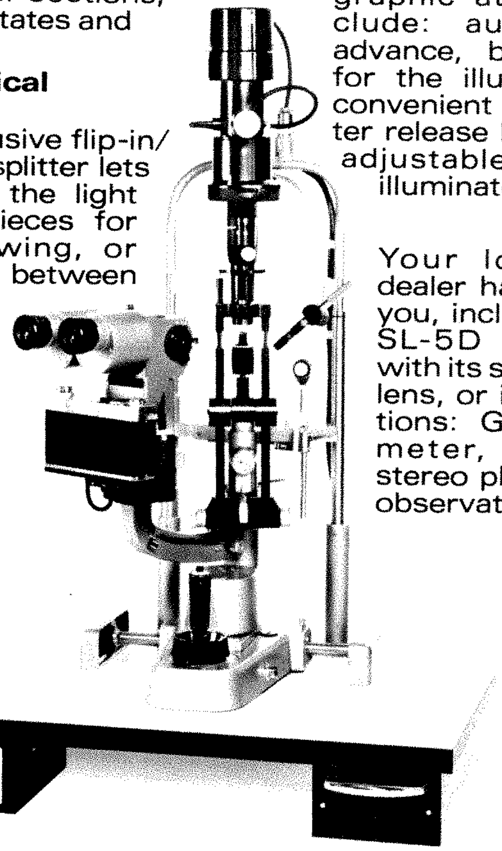

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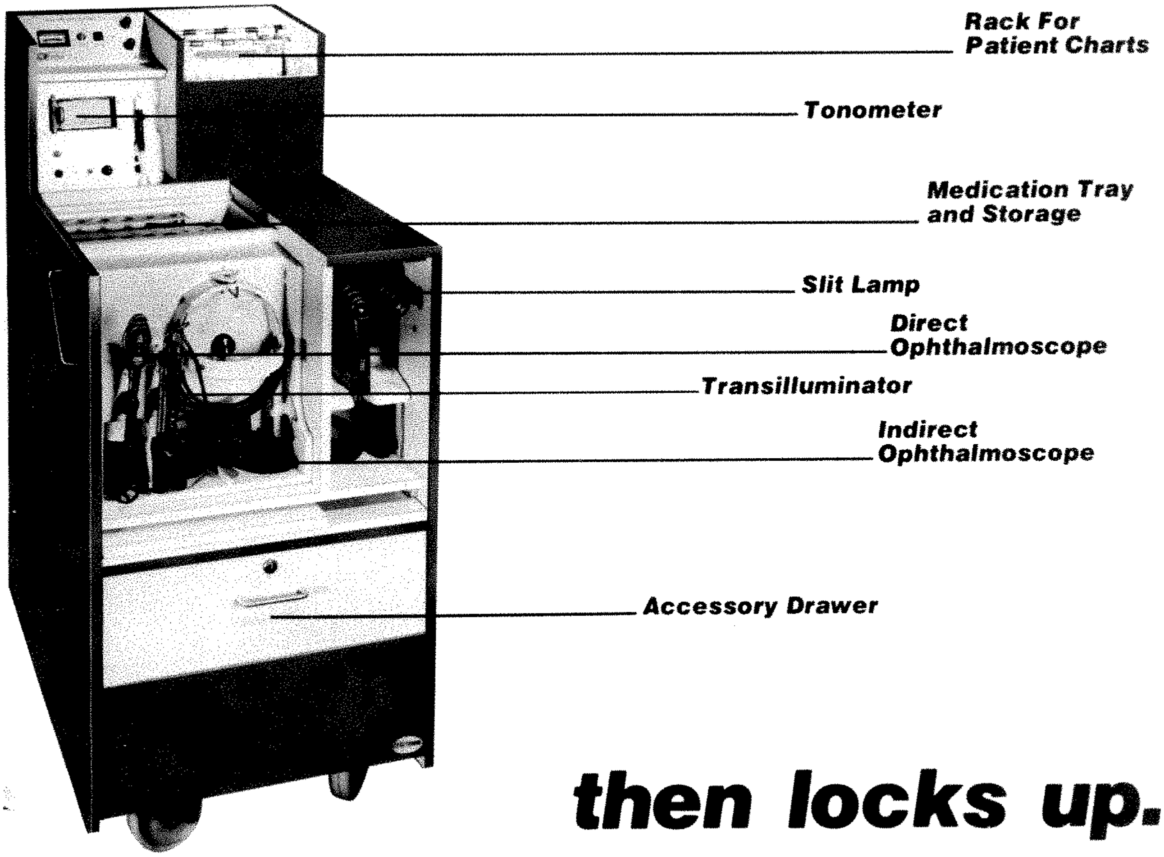

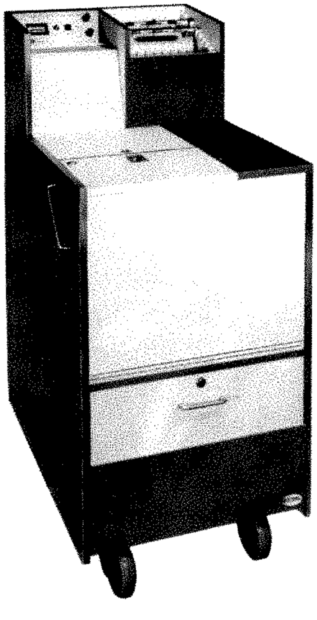

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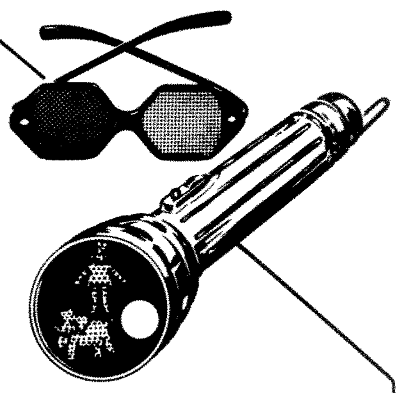

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Contraindications: 1. Active uveal inflammation.

2. Most cases of angle-closure glaucoma, due to the possibility of increasing angle block.

3. Hypersensitivity to the active or inactive ingredients.

Warnings: 1. *Use in Pregnancy:* Safe use of anticholinesterase medications during pregnancy has not been established, nor has the absence of adverse effects on the fetus or on the respiration of the neonate.

2. Succinylcholine should be administered only with great caution, if at all, prior to or during general anesthesia to patients receiving anticholinesterase medication because of possible respiratory or cardiovascular collapse.

3. Caution should be observed in treating glaucoma with PHOSPHOLINE IODIDE in patients who are at the same time undergoing treatment with systemic anticholinesterase medications for myasthenia gravis, because of possible adverse additive effects.

Precautions: 1. Gonioscopy is recommended prior to initiation of therapy.

2. Where there is a quiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and ciliary muscle contraction that may occur.

3. While systemic effects are infrequent, proper use of the drug requires digital compression of the nasolacrimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

4. Temporary discontinuance of medication is necessary if salivation, urinary incontinence, diarrhea, profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities occur.

5. Patients receiving PHOSPHOLINE IODIDE who are exposed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin. During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable.

6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer, pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.

7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.

8. PHOSPHOLINE IODIDE (echothiophate iodide) should be used with great caution, if at all, where there is a prior history of retinal detachment.

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2. Stinging, burning, lacrimation, lid muscle twitching, conjunctival and ciliary redness, browache, induced myopia with visual blurring may occur.

3. Activation of latent iritis or uveitis may occur.

4. Iris cysts may form, and if treatment is continued, may enlarge and obscure vision. This occurrence is more frequent in children. The cysts usually shrink upon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Rarely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative esotropia.

5. Prolonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.

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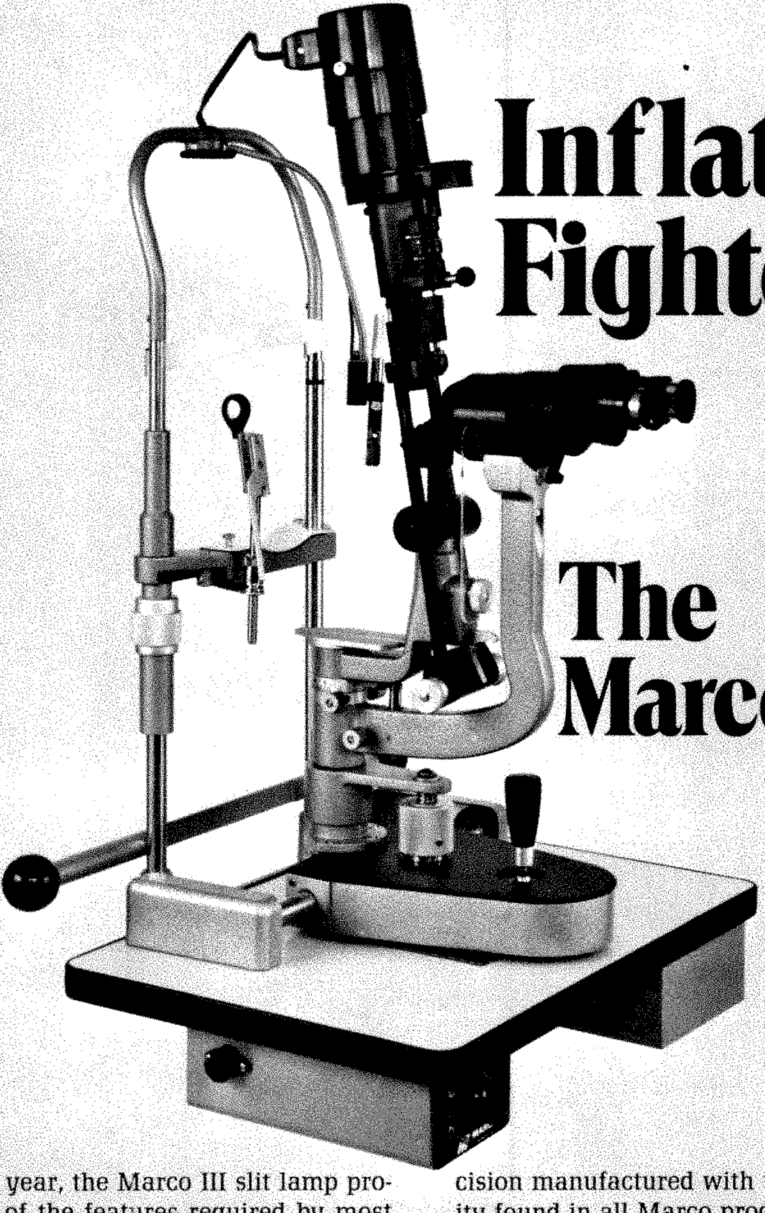

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For any further information, please contact Miss Katherine Sgabelloni, Coordinator Ophthalmic Services, Park Ridge Hospital, 1555 Long Pond Road, Rochester, New York 14626. Phone: (716) 225-7150, extension 4054.

St. Vincent's Hospital and Medical Center of New York

Pediatric Ophthalmology and Strabismus Update

October 4, 5 and 6, 1979

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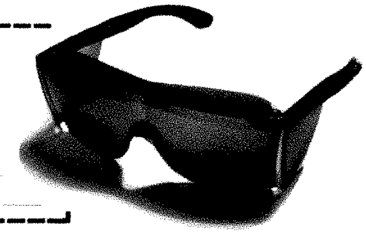

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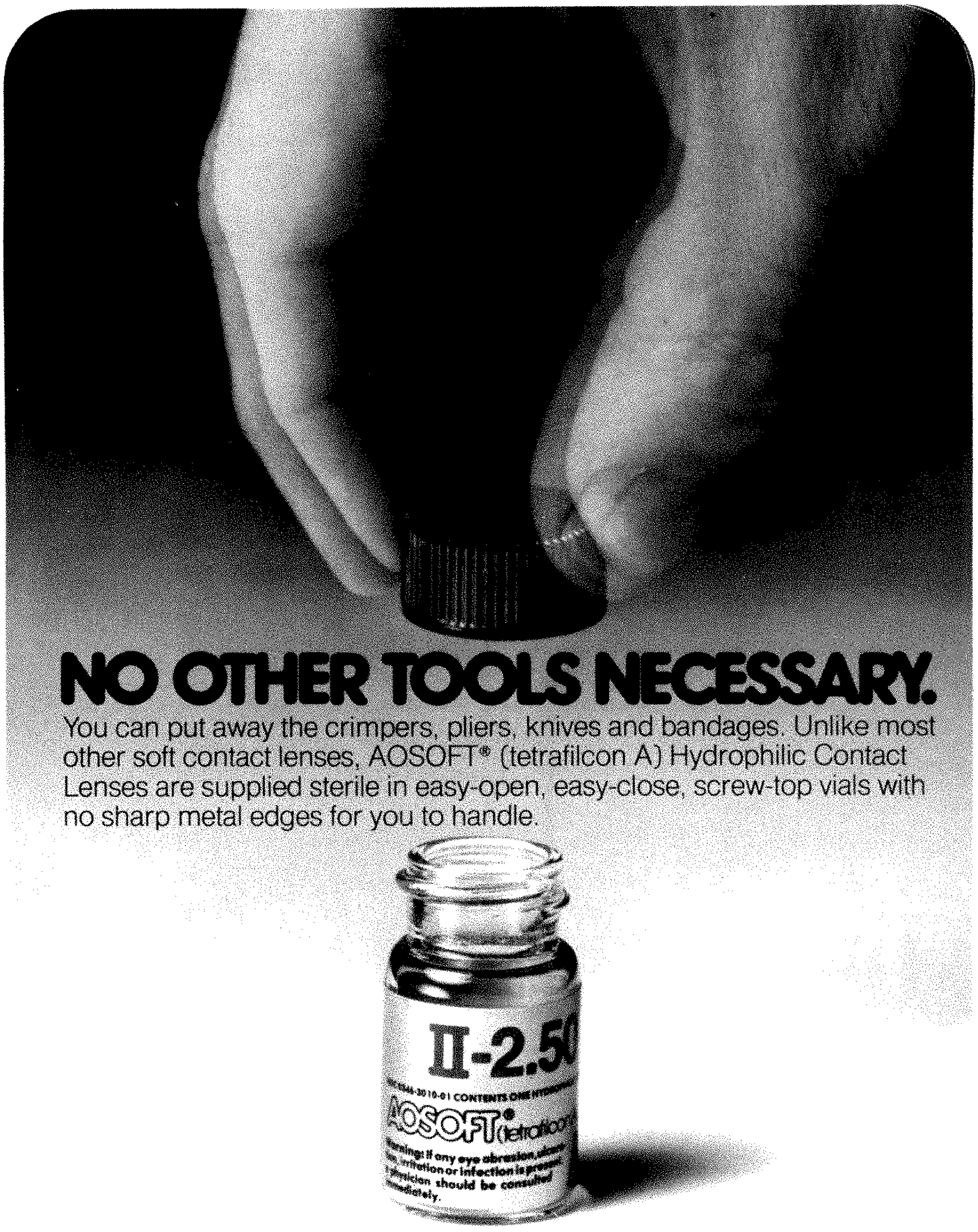
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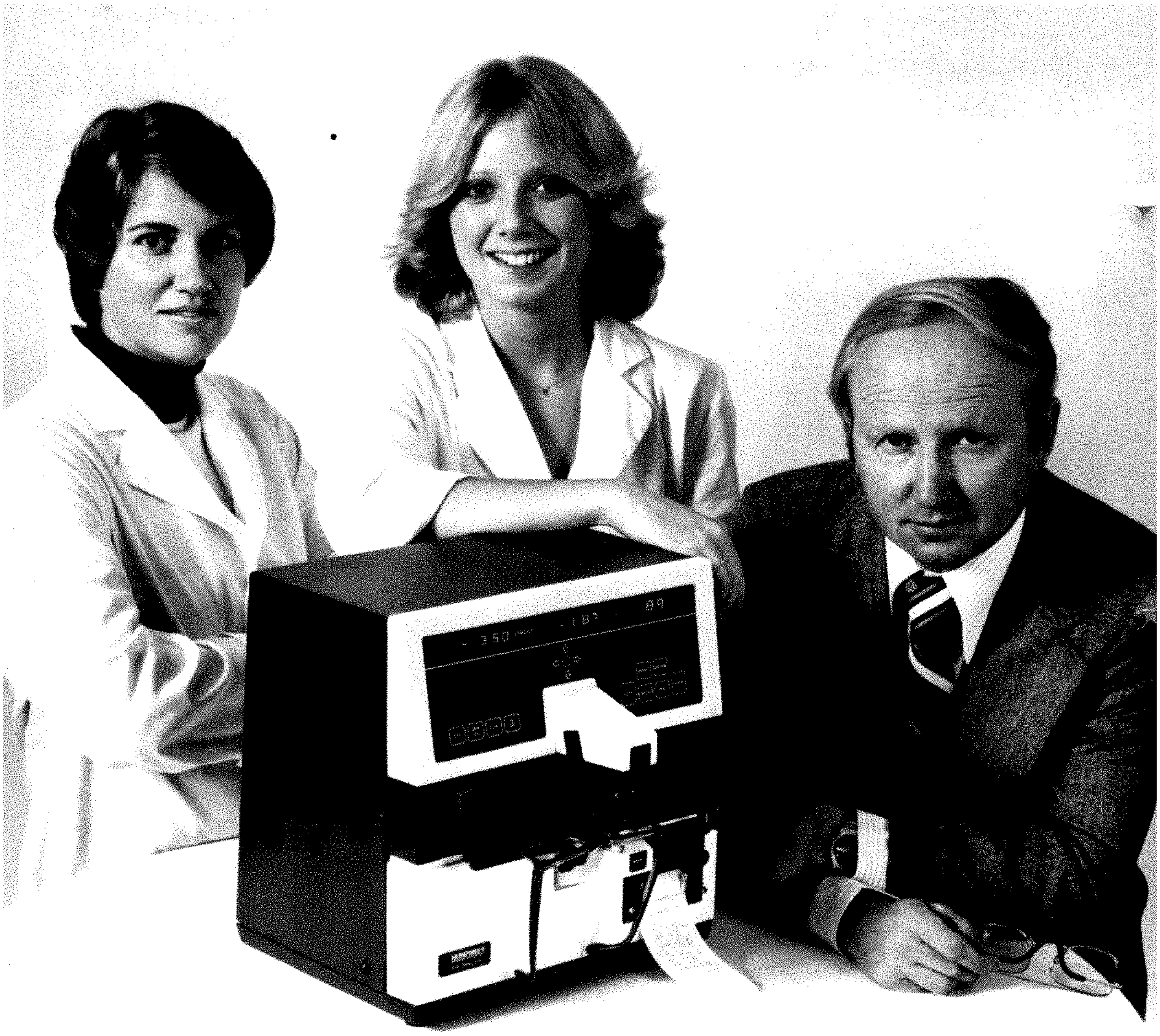

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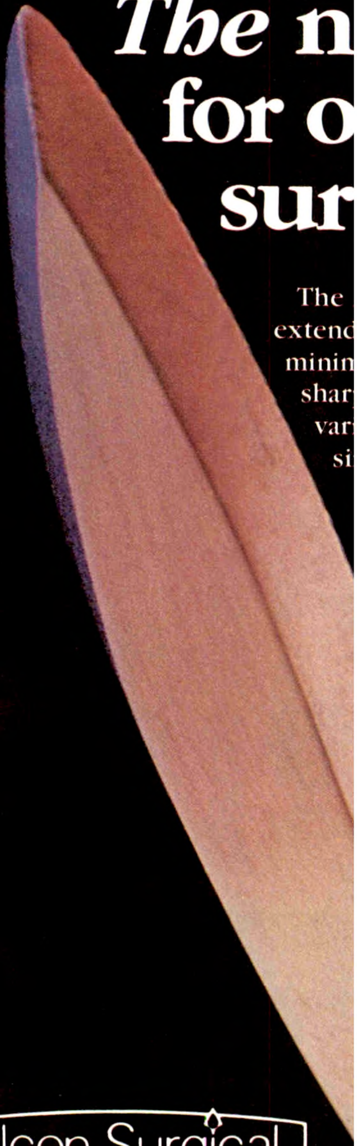

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Pain, photophobia, lacrimation and blepharospasm are usual presenting symptoms. Prompt institution of therapy is vital to avoid possible visual difficulty.

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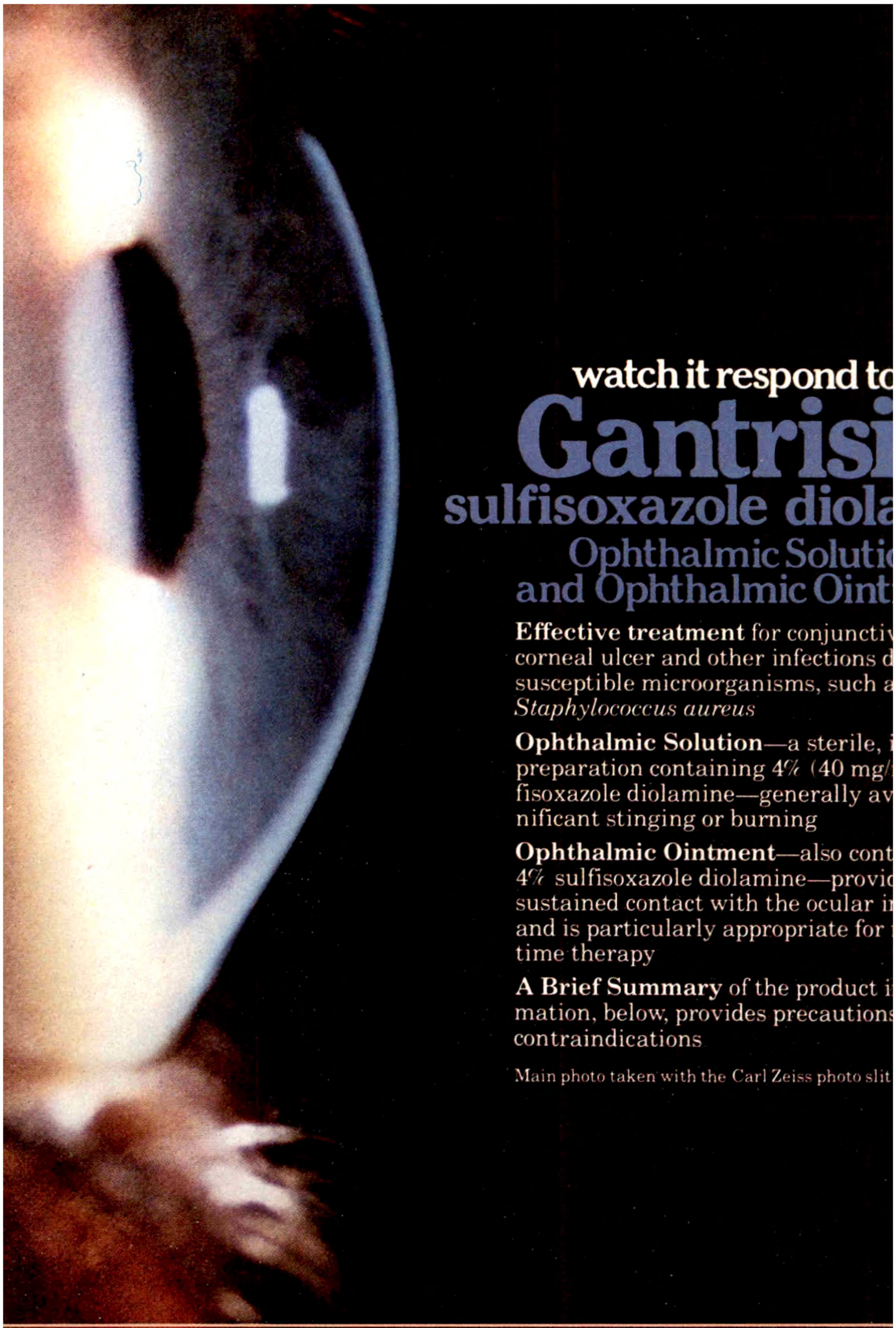

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Effective treatment for conjunctiv
corneal ulcer and other infections d
susceptible microorganisms, such a
Staphylococcus aureus

Ophthalmic Solution—a sterile, i
preparation containing 4% (40 mg/
fisoxazole diolamine—generally av
nificant stinging or burning

Ophthalmic Ointment—also cont
4% sulfisoxazole diolamine—provid
sustained contact with the ocular i
and is particularly appropriate for
time therapy

A Brief Summary of the product i
mation, below, provides precautions
contraindications

Main photo taken with the Carl Zeiss photo slit

Dosage and Administration: Solution: 2-3 drops in eye 3
or more times daily. Take care not to contaminate dropper.

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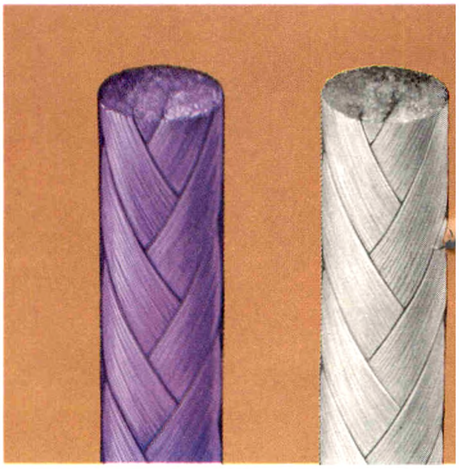

Available braided, dyed and undyed.

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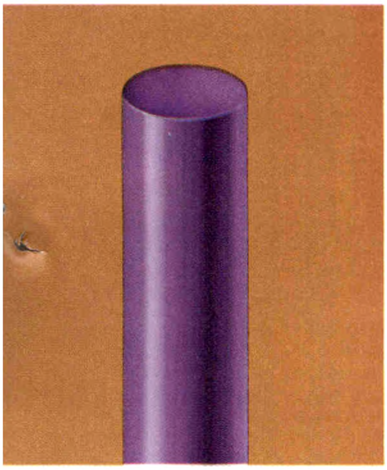

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(See next page for complete product information.)

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VICRYL*
(Polyglactin 910) suture

COMPLETE PRODUCT INFORMATION

VICRYL* (Polyglactin 910)
Synthetic Absorbable Suture

DESCRIPTION VICRYL (polyglactin 910) synthetic absorbable suture is prepared from a copolymer of glycolide and lactide. These substances are derived respectively from glycolic and lactic acids. The empirical formula of the copolymer is $(C\ H\ O\ )_m(C\ H_4O\ )_n$

VICRYL sutures are sterile, inert, nonantigenic, nonpyrogenic and elicit only a mild tissue reaction during absorption. The braided and monofilament sutures are colored violet to enhance visibility in tissue. The braided suture is also available undyed (natural).

ACTIONS Two important characteristics describe the *in vivo* behavior of absorbable sutures: first, tensile strength retention, and second, the absorption rate (loss of mass).

Subcutaneous tissue implantation studies of VICRYL suture in rats show at two weeks post-implantation approximately 55% of its original tensile strength remains, while at three weeks approximately 20% of its original strength is retained.

Intramuscular implantation studies in rats show that the absorption of VICRYL suture is minimal until about the 40th post-implantation day. Absorption is essentially complete between the 60th and 90th days.

INDICATIONS VICRYL synthetic absorbable suture is intended for use as an absorbable suture or ligature.

CONTRAINDICATIONS This suture, being absorbable, should not be used where extended approximation of tissues under stress is required.

WARNINGS The safety and effectiveness of VICRYL (polyglactin 910) suture in neural tissue, and in cardiovascular surgery have not been established.

Under certain circumstances, notably orthopedic procedures, immobilization by external support may be employed at the discretion of the surgeon.

Do not resterilize.

PRECAUTIONS VICRYL suture knots must be properly placed to be secure. Place the first throw in precise position for the final knot, using a double loop; tie the second throw square, using horizontal tension; additional throws are advisable.

Skin and conjunctival sutures remaining in place longer than 7 days may cause localized irritation and should be removed as indicated.

Acceptable surgical practice must be followed with respect to drainage and closure of infected wounds.

ADVERSE REACTIONS Reactions reported in clinical trials which may have been suture related have been minimal. These include skin redness and induration, rare instances of hemorrhage, anastomotic leakage, wound separation in the eye, and abscesses.

DOSAGE AND ADMINISTRATION Use as required per operation.

HOW SUPPLIED VICRYL sutures are available sterile, as braided dyed (violet) and undyed (natural) strands in sizes 3 to 8-0, in a variety of lengths, with and without needles, and on LIGAPAK* ligating reels. VICRYL sutures, monofilament, dyed (violet) are available in sizes 9-0 and 10-0, in a variety of lengths with needles.

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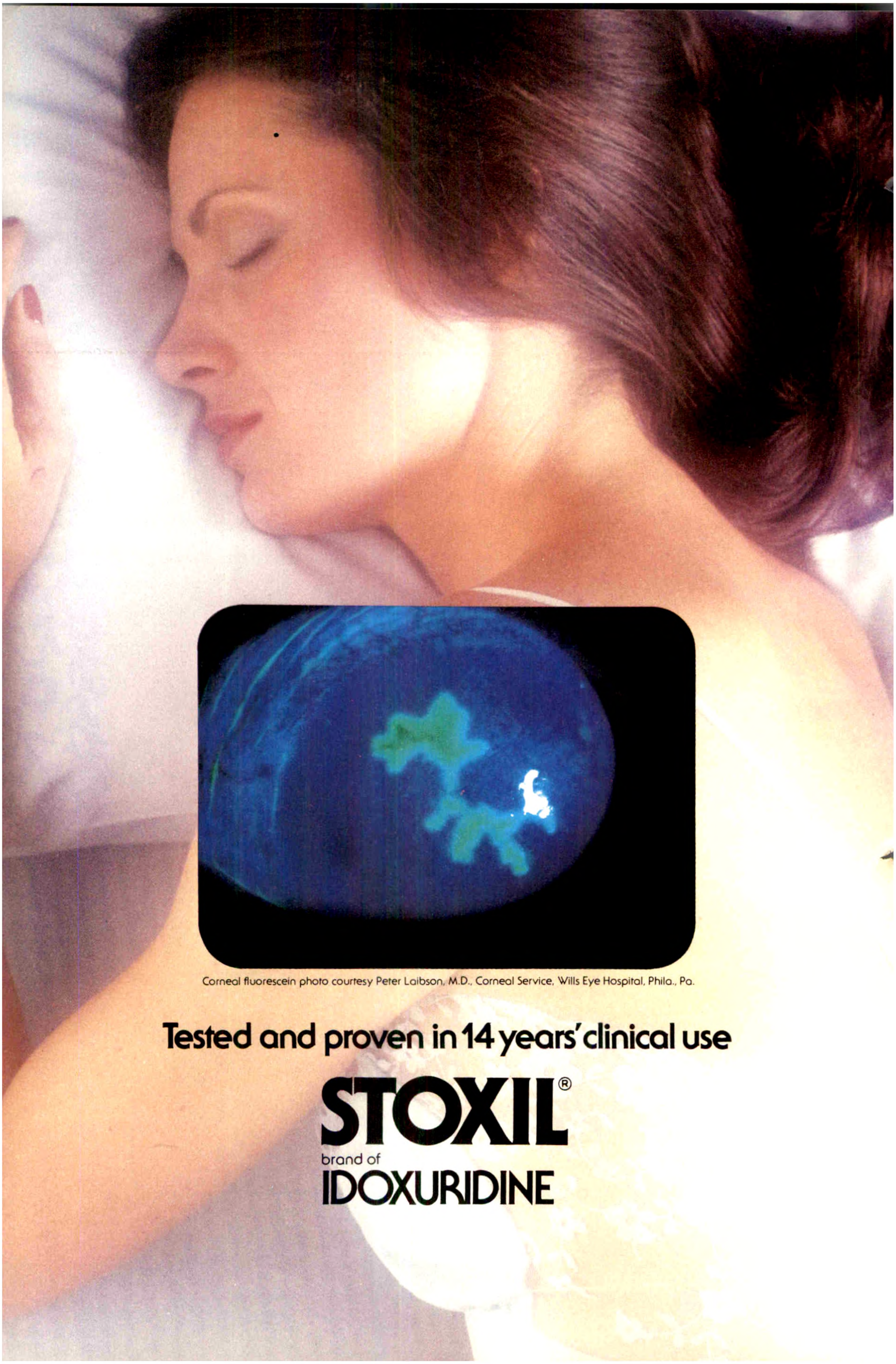

Corneal fluorescein photo courtesy Peter Laibson, M.D., Corneal Service, Wills Eye Hospital, Phila., Pa.

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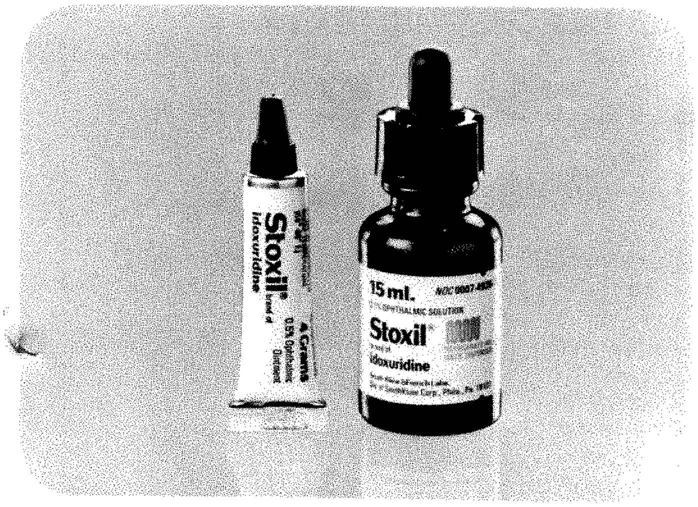

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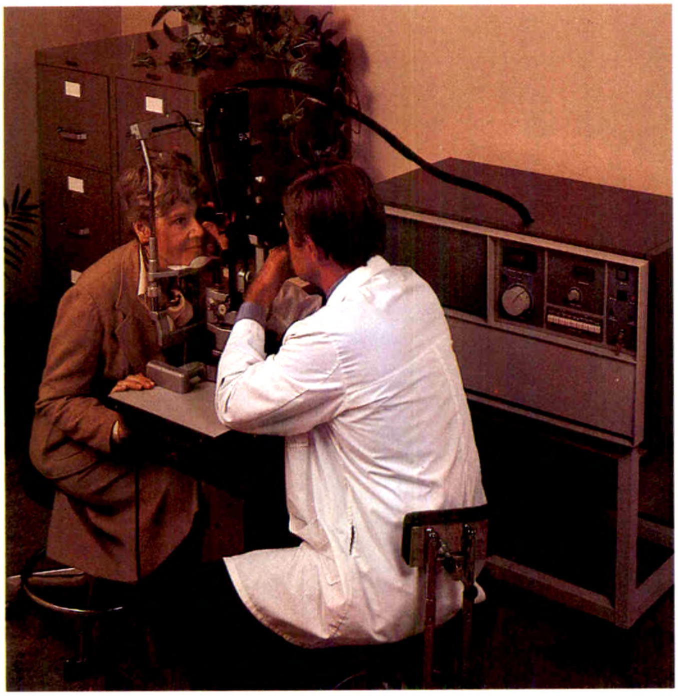

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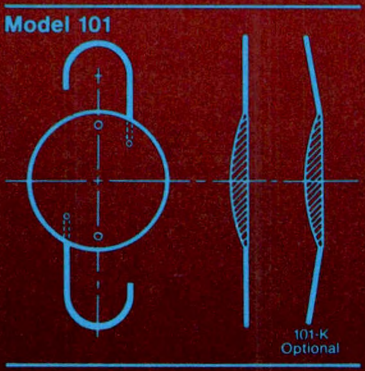

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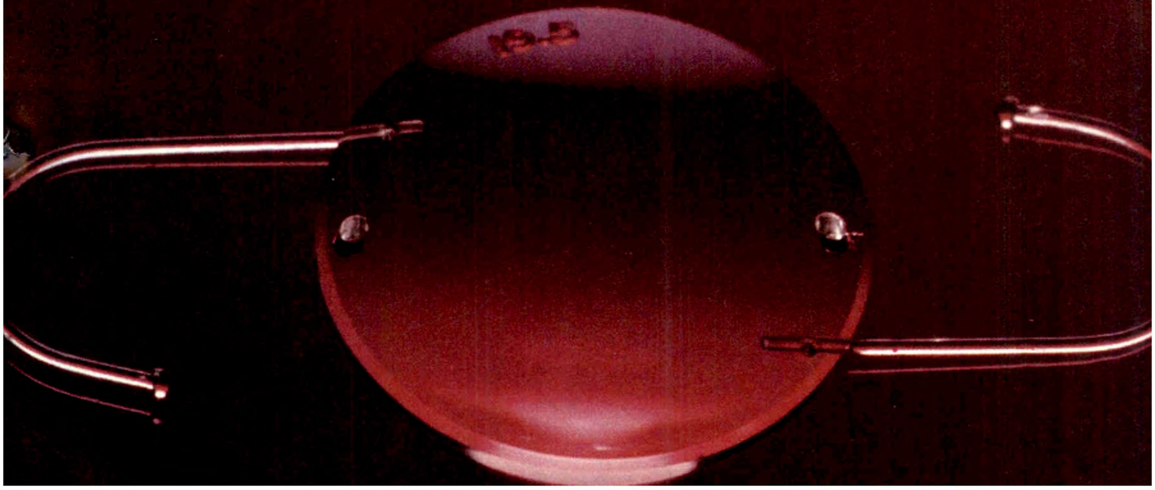

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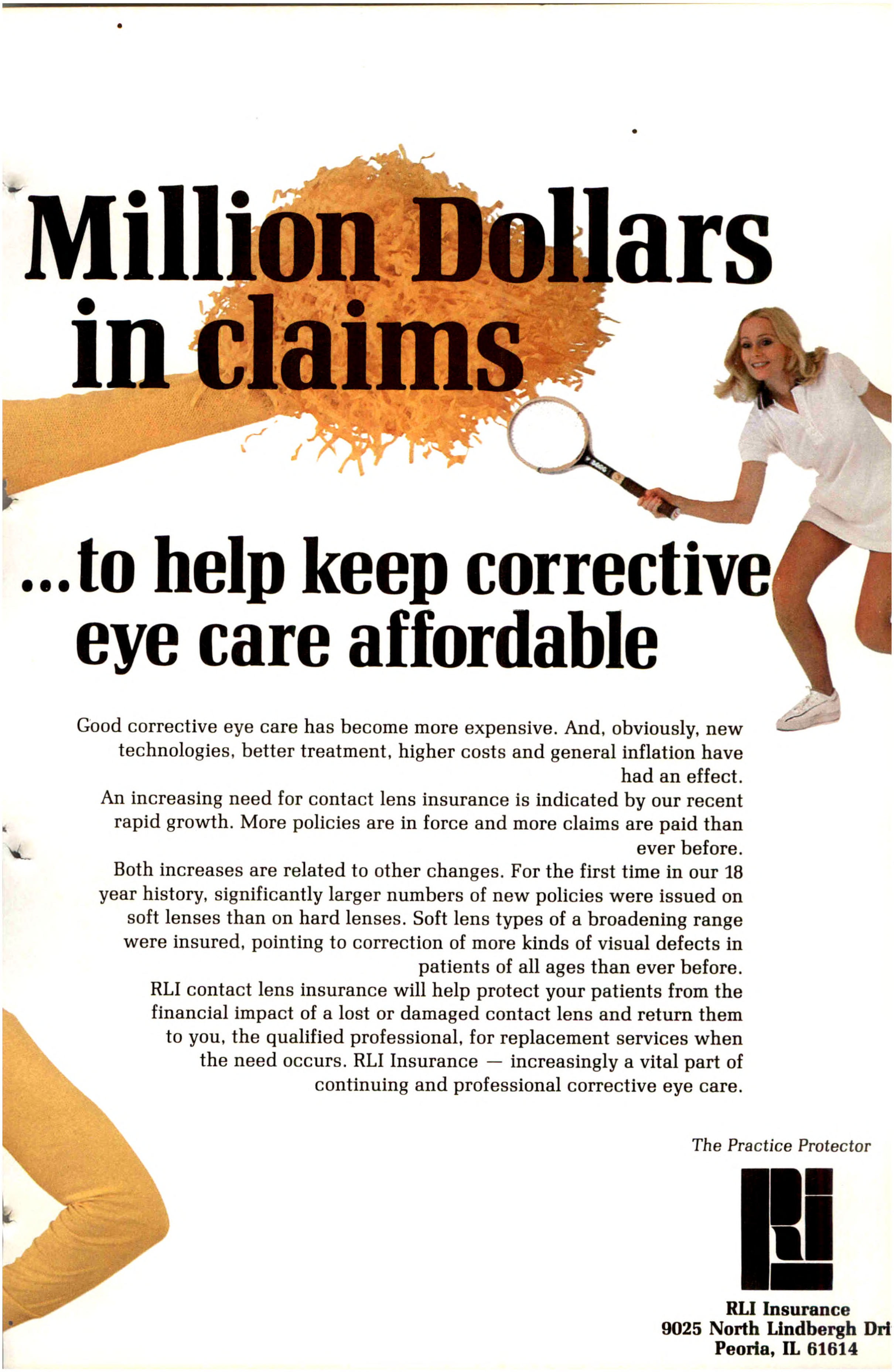

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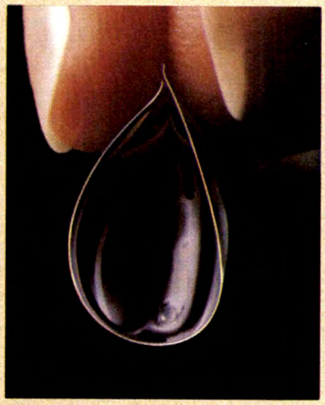

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[1] Malin, H. and Rorabaugh, D. "Fitting Small, Thin Hydrocurve Lenses." Contact Lens Forum, April, 1979.

[2] Available in 5° increments.

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HYDROCURVE II® (bufilcon A) Contact Lenses

(bufilcon A 55% water 45%)

DESCRIPTION

The HYDROCURVE II (bufilcon A) Contact Lens is a hemispherical shell of approximately 14 mm chord diameter and 0.1 to 1.0 mm thickness. The lens material, bufilcon A, is a hydrophilic random copolymer of 2-hydroxyethyl methacrylate, N-(1, 1-Dimethyl-3-oxobutyl)-acrylamide, and methacrylic acid. The polymer is a 3-dimensional network of copolymer chains joined by trimethylolpropane trimethacrylate crosslinks at a density of about one crosslink for every 1400 monomer units. It consists of 55% bufilcon A and 45% water when immersed in normal saline.

ACTIONS

In its hydrated state a HYDROCURVE II Contact Lens becomes soft and pliable. When placed on the human cornea the hydrated lens acts as a refracting medium to compensate spherical ametropias. The material has a refractive index of 1.43.

INDICATIONS

HYDROCURVE II Contact Lenses are indicated for the correction of visual acuity in persons with non-diseased eyes who have spherical ametropias, refractive astigmatism of 1.50 diopters or less and/or corneal astigmatism of 2.00 diopters or less and aphakia.

CONTRAINDICATIONS

HYDROCURVE II Contact Lenses are contraindicated by the presence of any of the following conditions:

1. Acute and subacute inflammations of the anterior segment of the eye.
2. Any eye disease which affects the cornea or conjunctiva.
3. Insufficiency of lacrimal secretion.
4. Corneal hypoesthesia.
5. Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Medicaments and Eye Drops

The HYDROCURVE II Contact Lens must be stored ONLY in BOILnSOAK® Sterile Preserved Saline Storage Solution or FLEXSOL® Sterile Solution for Disinfection and Storage. No ophthalmic solutions or medicaments, including conventional hard contact lens solutions, can be used by HYDROCURVE II Contact Lens wearers prior to or while the lens is in place on the eye. Only the recommended disinfectant soaking solutions BOILnSOAK or FLEXSOL, the recommended cleaners SOFT MATE® or PREFLEX®, NORMOL® Rinsing Solution or the recommended lubricating solution ADAPETTES® may be used on the HYDROCURVE II Contact Lens.

Abrasions and Infections

If the lens becomes less comfortable to the wearer than when it was first placed on the wearer's cornea, this may indicate the presence of a foreign body. The lens should be removed immediately and the patient examined. If any corneal abrasion, ulceration, irritation or infection is present, a physician should be consulted immediately.

Aphakic Patients

Aphakic patients should not be fitted with HYDROCURVE II Contact Lenses during the postoperative period until, in the opinion of the surgeon, the eye has healed completely.

Lens Care Regimen

Patients must adhere to the recommended daily lens care regimen of HYDROCURVE II Contact Lenses. Failure to follow this procedure may result in development of serious ocular infections.

Wearing Restrictions

The HYDROCURVE II Contact Lens should be removed before sleeping or swimming or in the presence of noxious and irritating vapors.

Visual Blurring

When visual blurring occurs the lens must be removed until the condition subsides.

PRECAUTIONS

Storage

HYDROCURVE II Contact Lenses must be stored ONLY in BOILnSOAK or FLEXSOL Solution. If left exposed to air, the lenses will dehydrate. If a lens dehydrates, it should be soaked ONLY in BOILnSOAK or FLEXSOL Solution until it returns to a soft, supple state.

Cleaning and Disinfection

HYDROCURVE II Contact Lenses must be BOTH cleaned and disinfected daily. One procedure does not replace the other. CLEANING is necessary to remove mucus and film from the lens surface. This can be accomplished by using either SOFT MATE or PREFLEX on a daily basis. Excessive deposits may damage the lens; therefore, if this occurs, evaluation of the lens care procedures should be made.

DISINFECTION with a HYDROCURVE Patient Disinfection Unit and BOILnSOAK Solution has been tested microbiologically and shown to be an effective disinfection procedure for HYDROCURVE II Contact Lenses.

HYDROCURVE II Contact Lenses must be cleaned daily with SOFT MATE or PREFLEX. Fresh BOILnSOAK Solution must be used daily for storing the lenses. The HYDROCURVE Carrying Case must be emptied and refilled with fresh BOILnSOAK Solution each time the lens is stored.

If a HYDROCURVE Patient Disinfection Unit is not available for disinfection of the lenses, the lenses must be boiled in their Carrying Case in a pan of water for 10 minutes.

CHEMICAL DISINFECTION with FLEXSOL has been tested microbiologically and shown to be an effective disinfection procedure for HYDROCURVE II Contact Lenses. HYDROCURVE II Contact Lenses must be cleaned and rinsed daily, or after wearing, with PREFLEX and NORMOL. The HYDROCURVE Twin Case must be emptied and refilled with fresh FLEXSOL prior to disinfecting the lenses. Fresh FLEXSOL must be used daily for storing and disinfecting the lenses.

WARNING

DO NOT MIX OR ALTERNATE THE DISINFECTION AND STORAGE SYSTEMS. FLEXSOL SHOULD NOT BE USED WITH HEAT.

Hygiene

Patients must wash and rinse hands thoroughly, and dry with a lint-free towel before handling the lenses.

Cosmetics, lotions, soaps and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Fluorescein

Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with normal saline solution and wait at least one hour before replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescein irreversibly.

ADVERSE REACTIONS

Serious corneal damage may result from wearing a lens which has been soaked in a conventional hard contact lens solution containing preservatives which should not be used with soft hydrophilic contact lenses.

Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritation.

Very rarely a lens may adhere to an eye as a result of the patient sleeping while wearing the lens, or wearing a hypotonic lens. If a lens adheres for any reason, patients may be instructed to apply a few drops of BOILnSOAK Solution or ADAPETTES and wait until the lens moves freely before removing the lens.

Rainbows or halos around objects or blurring of the vision may occur if the lenses are worn continuously for too long a time. Removal of the lenses and a rest period of at least one hour generally relieves these symptoms.

Excessive tearing, unusual eye secretions, and photophobia are not normal; if these symptoms occur, the patient should be examined to determine their cause.

DOSAGE and ADMINISTRATION

Conventional methods of fitting contact lenses do not apply to HYDROCURVE II (bufilcon A) Contact Lenses. For a detailed description of the fitting technique, refer to the HYDROCURVE Fitting Guide, copies of which are available from:

Soft Lenses, Inc.
8006 Engineer Road
San Diego, California 92111

WEARING SCHEDULE SHOULD BE DETERMINED BY THE EYE CARE PRACTITIONER. STUDIES HAVE NOT BEEN PERFORMED TO SUPPORT SAFETY FOR EXTENDED WEAR OF THIS LENS.

Day	Maximum Wearing Time (Continuous Hours)
1	3
2	3
3	4
4	4
5	6
6	6
7	8

There may be a tend
tient to overwear the
Therefore, the import
to the initial daily wea
should be stressed to

Care must be taken c
to assure that the pat
with a HYDROCURV
Kit and fully understa
handling instructions
As with any contact le
recall visits are neces
patient health and co
instructions.

HOW SUPPLIED

Each lens is supplied
vial containing sterile
solution. The glass vi
the base curve, diopt
eter and the manufac
ber of the lens.

To assure proper lens
dling, each HYDROC
MUST BE supplied w
HYDROCURVE Pati

The HYDROCURVE
Care Kit is a package
lens cleaning, disinfe
age, consisting of the

HYDROCURVE Carr
Case . . . .
HYDROCURVE Pati
Disinfection Unit . .
BOILnSOAK® Sterile
Preserved Saline
Storage Solution .
SOFT MATE® Sterile
(optional) or . . . . .
PREFLEX® Sterile Cl
(optional) . . . .
Patient Instructions .

Chemical Disinfectio
DROCURVE Patient
package required for
method of lens cleani
tion and storage cons
following:

PREFLEX® Sterile
Cleaner . . . .
NORMOL® Sterile Ri
Solution . . . .
FLEXSOL® Sterile So
For Disinfection
and Storage . . . . .
HYDROCURVE Twin
Case . . . .
Patient Instructions .

CAUTION: FEDERAL
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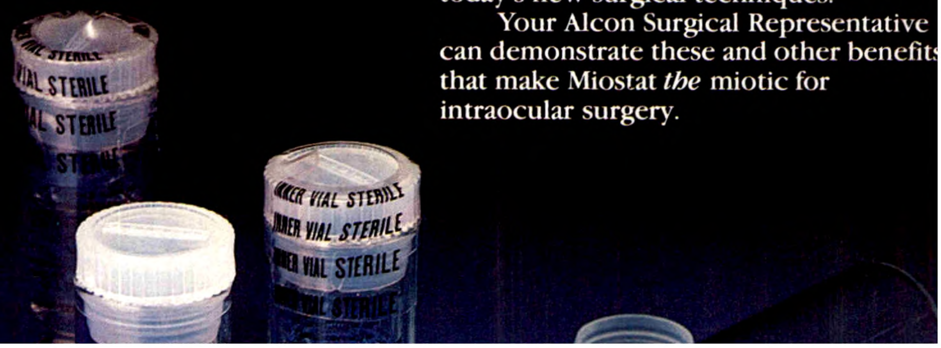

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- ☐ Duolube (formerly Muro #104 Petrolatum Ointment)
- ☐ Gonio-GEL

VIRA-A®
(vidarabine ophthalmic ointment), 3%

Description. Vira-A (vidarabine, adenine arabinoside, or Ara-A) is an antiviral drug for topical ophthalmic administration. The chemical name is 9-β-D-arabinofuranosyladenine. Each gram of the ophthalmic ointment contains 30 mg of vidarabine monohydrate equivalent to 28.11 mg of vidarabine in a sterile, inert, petrolatum base.

Clinical Pharmacology. Vira-A is a purine nucleoside obtained from fermentation cultures of *Streptomyces antibioticus*. Vira-A possesses *in vitro* and *in vivo* antiviral activity against Herpes simplex types 1 and 2, Varicella-Zoster, and Vaccinia viruses. Except for Rhabdovirus and Oncornavirus, Vira-A does not display *in vitro* antiviral activity against other RNA or DNA viruses, including Adenovirus.

The antiviral mechanism of action has not been established. Vira-A appears to interfere with the early steps of viral DNA synthesis. Vira-A is rapidly deaminated to arabinosylhypoxanthine (Ara-Hx), the principal metabolite. Ara-Hx also possesses *in vitro* antiviral activity but this activity is less than that of Vira-A. Because of the low solubility of Vira-A, trace amounts of both Vira-A and Ara-Hx can be detected in the aqueous humor only if there is an epithelial defect in the cornea. If the cornea is normal, only trace amounts of Ara-Hx can be recovered from the aqueous humor.

Systemic absorption of Vira-A should not be expected to occur following ocular administration and swallowing lacrimal secretions. In laboratory animals, Vira-A is rapidly deaminated in the gastrointestinal tract to Ara-Hx.

In contrast to topical idoxuridine, Vira-A demonstrated less cellular toxicity in the regenerating corneal epithelium of the rabbit.

Indications and Usage. Vira-A Ophthalmic Ointment, 3%, is indicated for the treatment of acute keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus types 1 and 2. Vira-A is also effective in superficial keratitis caused by Herpes simplex virus which has not responded to topical idoxuridine or when toxic or hypersensitivity reactions to idoxuridine have occurred. The effectiveness of Vira-A Ophthalmic Ointment, 3%, against stromal keratitis and uveitis due to Herpes simplex virus has not been established.

The clinical diagnosis of keratitis caused by Herpes simplex virus is usually established by the presence of typical dendritic or geographic lesions on slit-lamp examination. In controlled and uncontrolled clinical trials, an average of seven and nine days of continuous Vira-A Ophthalmic Ointment, 3%, therapy was required to achieve corneal re-epithelialization. In the controlled trials, 70 of 81 subjects (86%) re-epithelialized at the end of three weeks of therapy. In the uncontrolled trials, 101 of 142 subjects (71%) re-epithelialized at the end of three weeks. Seventy-five percent of the subjects in these uncontrolled trials had either not healed previously or had developed hypersensitivity to topical idoxuridine therapy.

The following topical antibiotics: gentamicin, erythromycin, chloramphenicol; or topical steroids: prednisolone or dexamethasone, have been administered concurrently with Vira-A Ophthalmic Ointment, 3%, without an increase in adverse reactions.

Normally, corticosteroids alone are contraindicated in Herpes simplex virus infections of the eye. If Vira-A Ophthalmic Ointment, 3%, is administered concurrently with topical corticosteroid therapy, corticosteroid-induced ocular side effects must be considered. These include corticosteroid-induced glaucoma or cataract formation and progression of a bacterial or viral infection.

Vira-A is not effective against RNA virus or adenoviral ocular infections. Vira-A is also not effective against bacterial, fungal, or chlamydial infections of the cornea or nonviral trophic ulcers.

Contraindication. Vira-A Ophthalmic Ointment, 3%, is contraindicated in patients who develop hypersensitivity reactions to it.

Warnings. ***Use in Pregnancy:*** Vira-A (vidarabine) parenterally is teratogenic in rats and rabbits. Ten percent Vira-A ointment applied to 10% of the body surface during organogenesis induced fetal abnormalities in rabbits. When 10% Vira-A ointment was applied to 2 to 3% of the body surface of rabbits, no fetal abnormalities were found. This dose greatly exceeds the total recommended ophthalmic dose in humans. The possibility of embryonic or fetal damage in pregnant women receiving Vira-A (vidarabine ophthalmic ointment), 3%, is remote. The topical ophthalmic dose is small, and the drug relatively insoluble. Its ocular penetration is very low. However, a safe dose for a human embryo or fetus has not been established. Consequently, Vira-A should be used only when clearly indicated.

It is not known whether Vira-A is excreted in human milk. As a general rule, because many drugs are excreted in human milk, nursing should not be undertaken while a patient is under treatment. However, excretion of vidarabine in breast milk is unlikely because Vira-A is rapidly deaminated in the gastrointestinal tract.

Mutagenic Potential: Results of *in vitro* experiments indicate that vidarabine can be incorporated into mammalian DNA and can induce mutation in mammalian cells (mouse L5178Y cell line). Thus far, *in vivo* studies have not been as conclusive, but there is some evidence (dominant lethal assay in mice) that vidarabine may be capable of producing mutagenic effects in male germ cells.

It has also been reported that vidarabine causes chromosome breaks and gaps when added to human leukocytes *in vitro*. While the significance of these effects in terms of mutagenicity is not fully understood, there is a well-known correlation between the ability of various agents to produce such effects and their ability to produce heritable genetic damage.

Oncogenic Potential: Chronic parenteral (IM) studies of vidarabine have been conducted in mice and rats.

In the mouse study, there was a statistically significant increase in liver tumor incidence among the vidarabine-treated females. In the same study, some vidarabine-treated male mice developed kidney neoplasia. No renal tumors were found in the vehicle-treated control mice or the vidarabine-treated female mice.

In the rat study, intestinal, testicular, and thyroid neoplasia occurred with greater frequency among the vidarabine-treated animals than in the vehicle-treated controls. The increases in thyroid adenoma incidence in the high-dose (50 mg/kg) males and the low-dose (30 mg/kg) females were statistically significant.

Hepatic megalocytosis, associated with vidarabine treatment, has been found in short- and long-term rodent (rat and mouse) studies. It is not clear whether or not this represents a preneoplastic change.

The recommended frequency and duration of administration should not be exceeded (see Dosage and Administration).

Precautions. The diagnosis of keratoconjunctivitis due to Herpes simplex virus should be established clinically prior to prescribing Vira-A Ophthalmic Ointment, 3%.

Patients should be forewarned that Vira-A Ophthalmic Ointment, 3%, like any ophthalmic ointment, may produce a temporary visual haze.

Although viral resistance to Vira-A has not been observed, this possibility may exist.

Adverse Reactions. Lacrimation, foreign-body sensation, conjunctival injection, burning, irritation, superficial punctate keratitis, pain, photophobia, punctal occlusion, and sensitivity have been reported with Vira-A Ophthalmic Ointment, 3%. The following have also been reported but appear disease-related: uveitis, stromal edema, secondary glaucoma, trophic defects, corneal vascularization, and hyphema.

Overdosage. Acute massive overdosage by oral ingestion of the ophthalmic ointment has not occurred. However, the rapid deamination to arabinosylhypoxanthine should preclude any difficulty. The oral LD_{50} for vidarabine is greater than 5,020 mg/kg in mice and rats. No untoward effects should result from ingestion of the entire contents of a tube.

Overdosage by ocular instillation is unlikely because any excess should be quickly expelled from the conjunctival sac. Too frequent administration should be avoided.

Dosage and Administration. Administer approximately one half inch of Vira-A Ophthalmic Ointment, 3%, into the lower conjunctival sac five times daily at three-hour intervals.

If there are no signs of improvement after 7 days, or complete re-epithelialization has not occurred by 21 days, other forms of therapy should be considered. Some severe cases may require longer treatment.

After re-epithelialization has occurred, treatment for an additional seven days at a reduced dosage (such as twice daily) is recommended in order to prevent recurrence.

How Supplied. N 0071-3677-07 (Stock 18-1677-139)

Vira-A Ophthalmic Ointment, 3%, is supplied sterile in ophthalmic ointment tubes of 3.5 g. The base is a 60:40 mixture of solid and liquid petrolatum. **UK**

PARKE-DAVIS
Division of Warner-Lambert Company
Morris Plains, NJ 07950

PD-JA-2320-1-P(5-78)

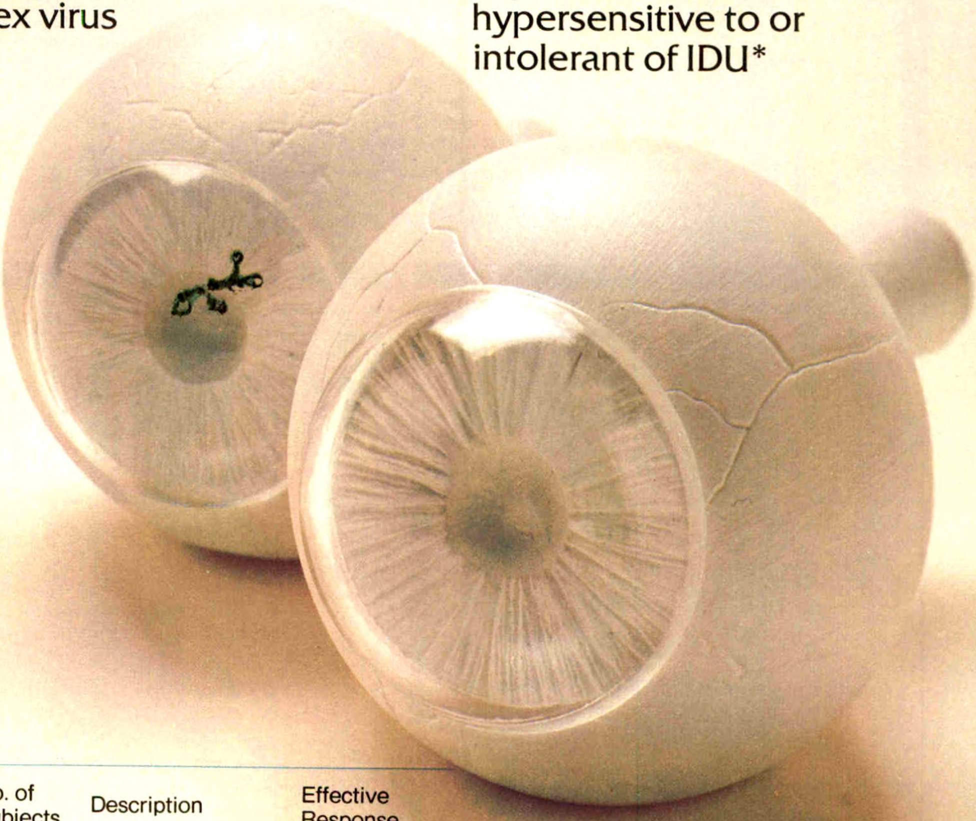
A LOGICAL FIRST CHOICE FOR HERPES SIMPLEX VIRUS KERATITIS

VIRA-A®

(vidarabine ophthalmic ointment), 3%

ot only as effective
s IDU in treating
erpes simplex virus
eratitis...

but is also effective
in patients resistant or
hypersensitive to or
intolerant of IDU*



inical Evidence	No. of Subjects	Description	Effective Response
ra-A ontrolled Trials	81	Previously untreated	70 subjects **(86%)**
ra-A ncontrolled Trials	142	75% had been resistant or hypersensitive to IDU	101 subjects **(71%)**

OTE: In the controlled and uncontrolled trials, 70 and 101 subjects, spectively, re-epithelialized at the end of three weeks on Vira-A.

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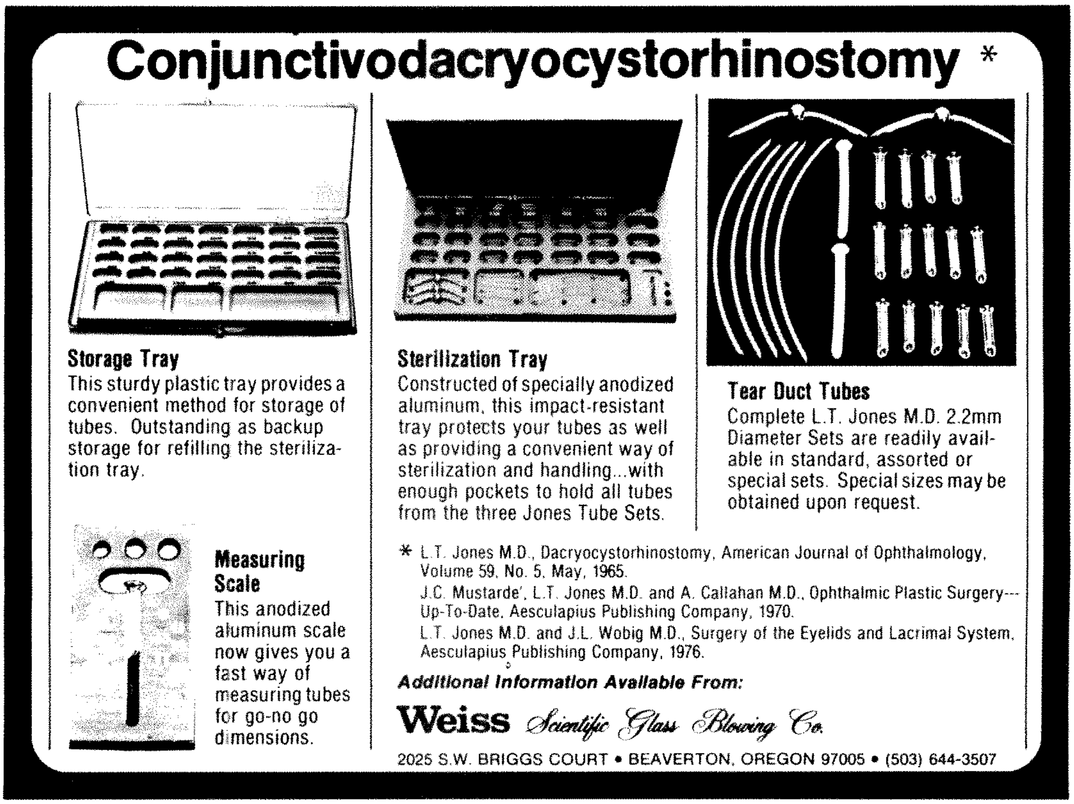
ta on file, Medical Department, Parke-Davis

ease see prescribing information on preceding page.

Vira-A is preservative-free.

PARKE-DAVIS



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* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
J.C. Mustarde', L.T. Jones M.D. and A. Callahan M.D., Ophthalmic Plastic Surgery—Up-To-Date, Aesculapius Publishing Company, 1970.
L.T. Jones M.D. and J.L. Wobig M.D., Surgery of the Eyelids and Lacrimal System, Aesculapius Publishing Company, 1976.

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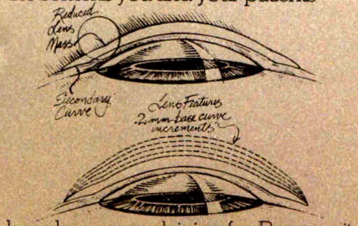
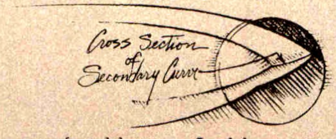


It's in the design. The bi-curve posterior surface with a relatively flat peripheral curve allows the GELFLEX Contact Lens to adapt to both the cornea and sclera. The secondary curve minimizes scleral impingement and promotes adequate tear flow through a tear-pump action for comfortable wear. And the lenticular anterior surface allows reduced lens mass, optimum flange contour and edge thickness, and better uniformity of thickness throughout the lens.



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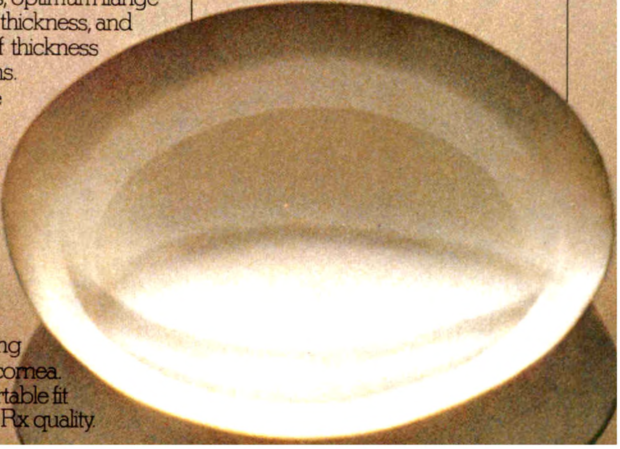

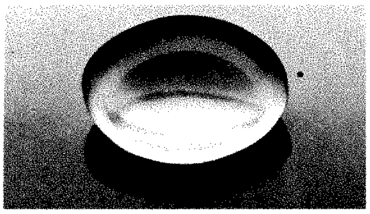

GELFLEX®(dimefilcon A) Contact Lenses

Description

The GELFLEX® (dimefilcon A) Contact Lens is a hemispherical shell of approximately 13 4mm chord diameter and 0.06mm to 0.80mm thickness. The lens material, dimefilcon A, is a hydrophilic copolymer of 2-hydroxethyl methacrylate and methyl methacrylate, cross-linked with triethylene glycol dimethacrylate. It consists of 65% dimefilcon A and 36% water by weight when immersed in normal saline solution buffered with sodium bicarbonate

Actions

Hydrated dimefilcon A material has a refractive index of 1 43. When placed on the human cornea, the GELFLEX® (dimefilcon A) Contact Lens acts as a refracting medium to correct for spherical ametropias

Indications

GELFLEX® (dimefilcon A) Contact Lenses are indicated for the correction of vision in persons with non-diseased eyes having spherical ametropias, refractive astigmatism of 1.50 diopters or less and/or corneal astigmatism of 2.00 diopters or less, and aphakia

Contraindications

GELFLEX® (dimefilcon A) Contact Lenses are contraindicated by the presence of any of the following conditions

1. Acute and subacute inflammations of the anterior segment of the eye,

2. Any eye disease which affects the cornea or conjunctiva,

3. Insufficiency of lacrimal secretion,

4. Corneal hypoesthesia,

5. Any systemic disease which may affect the eye adversely or may be exaggerated by wearing of contact lenses

Warnings

Medicaments and eye drops

GELFLEX® (dimefilcon A) Contact Lenses must be stored ONLY in Soft-Therm® or BOILnSOAK,® a sterile preserved saline solution, or in Flexsol,® a sterile soaking solution No ophthalmic solutions or medicaments, including conventional (hard) contact lens products, may be used by GELFLEX® (dimefilcon A) Contact Lens wearers immediately prior to or while the lens is worn Only the recommended disinfectant soaking solutions Soft-Therm,® BOILnSOAK® or Flexsol,® the recommended cleaners Soft Mate® or Preflex,® the recommended rinsing solution Normol,® or the recommended lubricating solution Adapettes,® may be used on the GELFLEX® (dimefilcon A) Contact Lens

Aphakic patients

Aphakic patients should not be fitted with a GELFLEX® (dimefilcon A) Contact Lens until, in the opinion of the surgeon, the eye has healed completely

Abrasions and infections

If a lens becomes less comfortable to the wearer than when it was first placed on the cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any corneal abrasion, ulceration, irritation or infection is present, a physician should be consulted immediately

Wearing restrictions

GELFLEX® (dimefilcon A) Contact Lenses should be removed before sleeping or swimming or in the presence of noxious and irritating vapors

Visual blurring

When visual blurring occurs, the lens must be removed until the condition subsides.

Lens care procedures

Patients must adhere to recommended daily lens care procedures. Failure to follow these procedures may result in development of serious ocular infections Before handling the lenses, hands must be washed, rinsed thoroughly, and dried with a lint-free towel. Cosmetics, lotions, soaps and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the lids must be kept closed until the hair spray has settled or the lenses should be inserted after the use of any sprays 'Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored Whenever fluorescein is used, irrigate the eyes with irrigating solution or preserved saline solution and wait at least one hour before replacing the lenses Too early replacement may allow the lenses to absorb residual fluorescein irreversibly

Precautions

Storage

GELFLEX® (dimefilcon A) Contact Lenses must be stored ONLY in Soft-Therm,® BOILnSOAK,® or Flexsol® If left exposed to air, the lenses will dehydrate, becoming hard and more prone to damage If a lens dehydrates, it should be soaked ONLY in Soft-Therm,® BOILnSOAK,® or Flexsol® until it returns to a soft, supple state Rehydration rarely takes more than 30 minutes after which the lenses should be cleaned and disinfected

Cleaning and disinfection

GELFLEX® (dimefilcon A) Contact Lenses MUST be BOTH cleaned and disinfected daily One procedure does not replace the other

Cleaning

Daily cleaning is necessary to remove mucus and other deposits from the lens surfaces. This is accomplished by using either Soft Mate® or Preflex® on a daily basis. Surface accumulations which are not removed daily may damage a lens If this should occur, reinstruct the patient in proper lens care procedures

Disinfection

Lenses must be disinfected daily according to the heat disinfection or chemical disinfection regimen

Heat disinfection with the GELFLEX® Disinfector, Contact Lens Case and Soft-Therm® or BOILnSOAK® has been tested microbiologically and shown to be an effective disinfection procedure for GELFLEX® (dimefilcon A) Contact Lenses

GELFLEX® (dimefilcon A) Contact Lenses must be cleaned daily with Soft Mate® or Preflex® Fresh Soft-Therm® or BOILnSOAK® must be used daily for storing the lenses

The GELFLEX® Contact Lens Case must be emptied and refilled with fresh Soft-Therm® or BOILnSOAK® solution each time the lenses are stored If a GELFLEX® Disinfector is not available for disinfection of the lenses, the lenses must be boiled in the GELFLEX® Contact Lens Case in a pan of water for 10 minutes.

Chemical disinfection with Flexsol® has been tested microbiologically and shown to be an effective disinfection procedure for GELFLEX® (dimefilcon A) Contact Lenses GELFLEX® (dimefilcon A) Contact Lenses must be cleaned and rinsed daily, or after wearing with Preflex® and Normol® The Burton, Parsons storage case must be emptied and refilled with fresh Flexsol® prior to disinfecting the lenses Fresh Flexsol® must be used daily for storing and disinfecting the lenses.

Warning

● Do not mix or alternate the disinfection and storage systems.
● Flexsol® should not be used with heat.

Adverse reactions

Serious corneal damage may result from wearing a lens which has been soaked in a conventional hard contact lens solution containing preservatives which should not be used with soft hydrophilic contact lenses.

On rare occasions, a lens may adhere to an eye as a result of the patient sleeping with the lens on or wearing a hypotonic lens If a lens adheres for any reason, apply a few drops of Soft-Therm® or BOILnSOAK® or Adapettes® directly to the eye and wait until the lens moves freely before removing it

The appearance of rainbows or halos around artificial lights, often symptomatic of corneal edema, or the blurring of vision may occur if lenses are worn for excessively long periods. Removal of the lenses for at least one hour will usually relieve these symptoms If symptoms persist, reevaluation should be made

Excessive tearing, unusual eye secretions and photophobia are not normal If these symptoms occur, the patient should be examined to determine their cause

Dosage and administration

Conventional methods of fitting hard contact lenses do not apply to fitting GELFLEX® (dimefilcon A) Contact Lenses For a detailed description of the fitting technique, refer to the GELFLEX® Fitting Guidelines – copies are available from Dow Corning Ophthalmics, Inc , 3556 Lexington Ave , El Monte, CA 91734

Day	Maximum Wearing Time (Continuous Hours)	Day	Maximum Wearing Time (Continuous Hours)
1	3	8	8
2	3	9	8
3	4	10	10
4	4	11	12
5	6	12	14
6	6	13	15
7	8	14	all waking hours

Wearing schedule should be determined by the eye care practitioner. Studies have not been performed to support safety for extended wear of this lens.

There may be a tendency for the patient to overwear the lenses initially Consequently, the importance of adhering to the initial daily wearing schedule should be stressed to the patient

At the time the lenses are dispensed, the patient MUST be provided with the GELFLEX® Patient Care Kit and printed instructional material There must also be assurance that the patient fully understands all care and handling procedures for the lenses

Each lens is supplied sterile in sealed glass vial containing sterile buffered saline solution The vial is marked with the base curve, dioptric power, diameter, expiration date and the manufacturing lot number of the lens

The GELFLEX® Patient Care Kit is a package required for lens cleaning, disinfection and storage of the lenses. It consists of the following:

Heat disinfection

GELFLEX® Disinfector

GELFLEX® Contact Lens Case

Soft-Therm® Sterile Preserved Saline Storage Solution (optional) or

BOILnSOAK® Sterile Preserved Saline Storage Solution (optional)

Soft Mate® Sterile Cleaner (optional) or

Preflex® Sterile Cleaner (optional)

Patient Instruction Booklet

The following GELFLEX® Patient Care Kit is a package required for the alternate method of cleaning storing and disinfecting GELFLEX® (dimefilcon A) Contact Lenses.

Chemical disinfection

Burton, Parsons Carrying and Storage Case for Chemical Disinfection

Preflex® Sterile Cleaner

Flexsol® Sterile Soaking Solution

Normol® Sterile Rinsing Solution

Adapettes® Sterile Lubricating Solution

Patient Instruction Booklet

CAUTION: Federal law prohibits dispensing without a prescription.

Soft-Therm® and Soft Mate® are made by Barnes-Hind Pharmaceuticals, Inc , Sunnyvale, CA 94086

BOILnSOAK,® Preflex,® Flexsol,® Normol® and Adapettes® are made by Burton, Parsons & Co , Inc , Washington, D C 20027

All items mentioned above are available in retail drug outlets.

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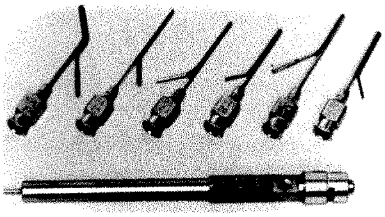

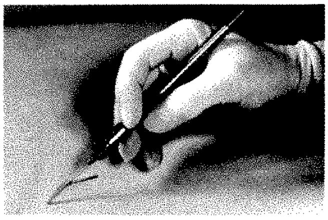

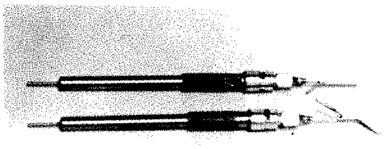

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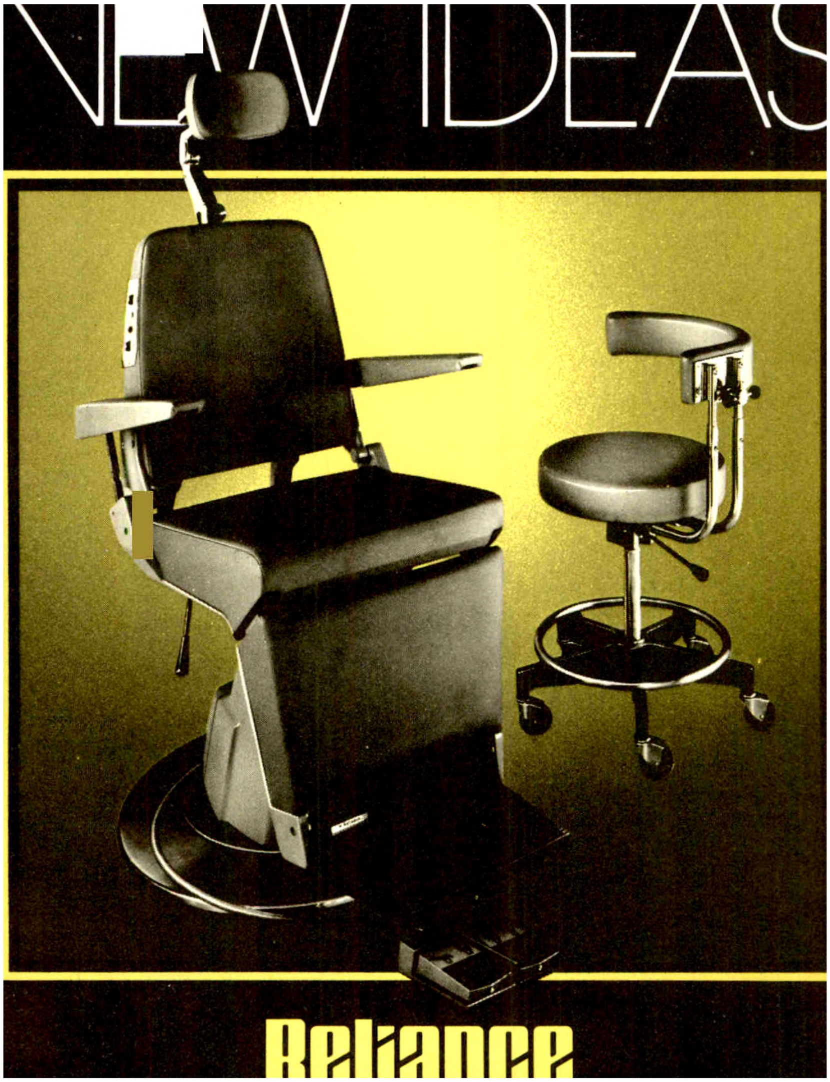
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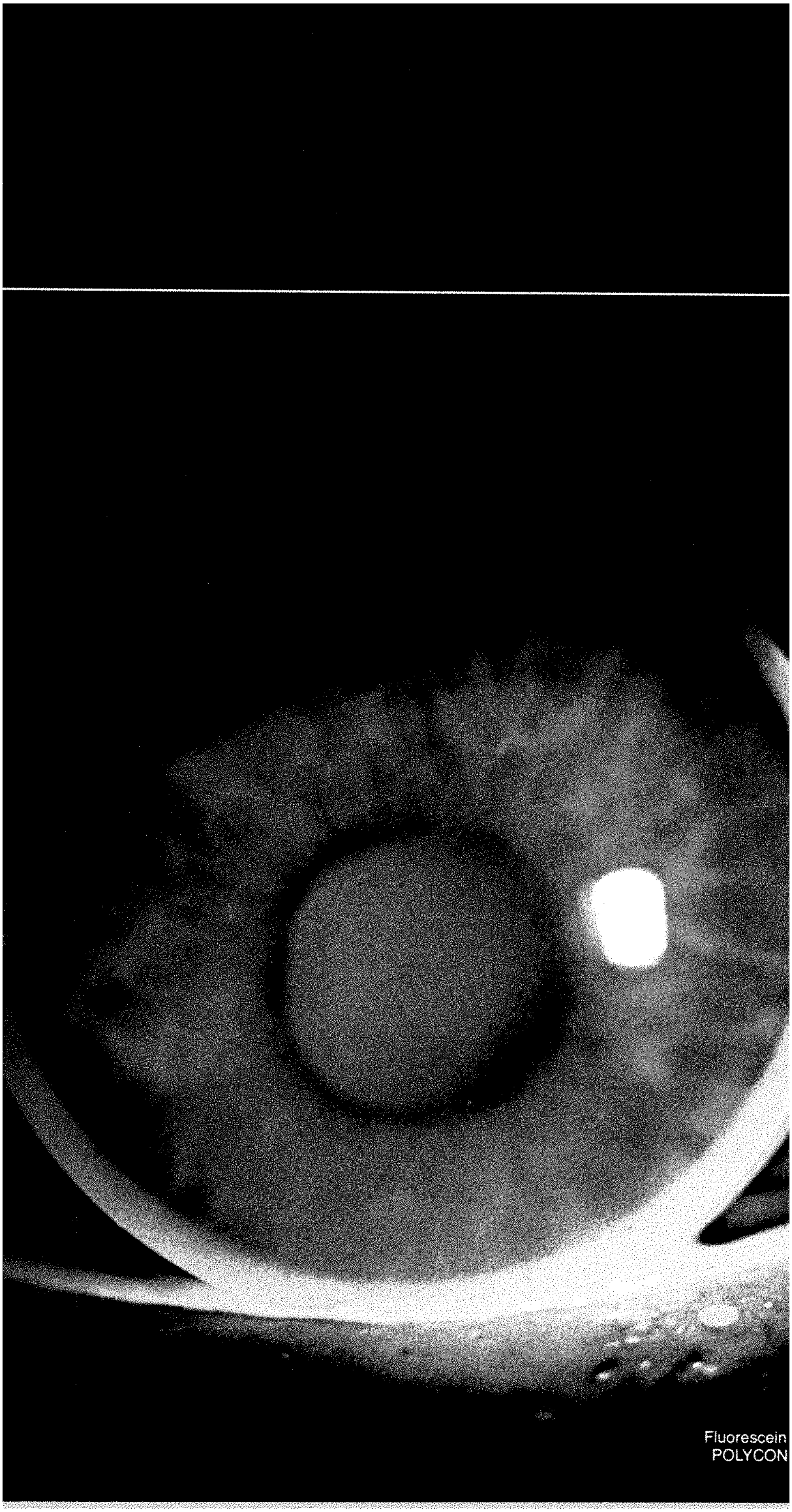
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POLYCON®
(silafocon A) CONTACT LENS

BRIEF SUMMARY

INDICATIONS
The POLYCON contact lens is indicated for the correction of visual acuity for persons with nondiseased eyes having ametropias, including those with high degrees of corneal or refractive astigmatism, as well as aphakia.

CONTRAINDICATIONS
Acute and subacute inflammations of the anterior segment of the eye; any eye disease which affects the cornea or conjunctiva; insufficiency of lacrimal secretion; corneal hyposthesia; or any systemic disease which may affect the eye or be exacerbated by wearing contact lenses.

WARNINGS
Abrasions and Infections: If the lens becomes less comfortable to the wearer than when first placed on the wearer's cornea, a foreign body may be present. The patient should remove the lens immediately. If discomfort persists, an eye care practitioner should examine the patient as soon as possible to determine if any corneal abrasion, ulceration, irritation or infection is present.

Aphakic Patients: Aphakic patients should not be fitted during the postoperative period until the surgeon determines that the eye has healed completely.

Lens Care Regimen: Only use Preflex® Sterile Cleaning Solution, Normol® Sterile Solution, and Adapettes® Sterile Lubricating Solution with the POLYCON lens. Store ONLY in Normol® Sterile Solution. Patients must adhere to recommended daily lens care regimen. FAILURE TO DO SO MAY RESULT IN DEVELOPMENT OF SERIOUS OCULAR INFECTIONS.

Medicaments and Eye Drops: Wearers must not use any medicaments or ophthalmic solutions other than Adapettes solution prior to inserting the lens or while the lens is on the eye.

Wearing Restrictions: Remove before sleeping or in the presence of noxious and irritating vapors.

PRECAUTIONS

CLEANING AND DISINFECTION
THE **POLYCON** CONTACT LENS MUST BE BOTH CLEANED AND DISINFECTED DAILY. ONE PROCEDURE DOES NOT REPLACE THE OTHER.

To disinfect the lens, it is stored in fresh Normol solution for a minimum of four hours daily in the patient lens storage case.

After removal of the lens from the storage case, it is rinsed thoroughly with Normol solution. Adapettes lubricating solution is applied to the lens surfaces as a wetting agent prior to inserting the lens in the eye.

ADVERSE REACTIONS
Rainbows or halos around objects or blurring of the vision. Excessive tearing, unusual eye secretions and photophobia.

FITTING AND WEARING SCHEDULE
Conventional methods of fitting contact lenses apply to the POLYCON contact lens.

NOTE: STUDIES HAVE NOT BEEN PERFORMED TO SUPPORT SAFETY OF THIS LENS FOR LONGER THAN THE RECOMMENDED WEARING TIME.

A POLYCON Contact Lens Patient Care Kit must be provided at the dispensing visit.

Preflex, Normol and Adapettes are trademarks of Burton, Parsons & Company, Inc.

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SYNTEX OPHTHALMICS, INC.
PHOENIX, ARIZONA 85015

ADSORBOCARPINE®
pilocarpine HCl in Adsorbobase®

Description: Adsorbocarpine Ophthalmic Solutions are sterile, buffered, stabilized solutions of pilocarpine HCl (U.S.P.) in Adsorbobase — (a combination of high molecular weight, water soluble polymers and povidone). Adsorbocarpine is available in three strengths: 1%, 2% and 4%.
All strengths, in addition to the respective amount of pilocarpine HCl, contain:

Vehicle: Adsorbobase and Hydroxyethylcellulose

Preservatives:	Benzalkonium Chloride	0.004%
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Indications: For the control of intraocular pressure; as a miotic in the treatment of chronic, simple, open-angle glaucoma; to counteract the effect of cycloplegics. The choice of strength should be determined by the severity of the condition and the response of the patient. To relieve tension in the treatment of acute glaucoma Adsorbocarpine may be used alone — prior to emergency surgery — or in combination with other miotics or carbonic anhydrase inhibitors.

Administration: The choice of concentration and the frequency of instillation should be determined by the severity of the condition and the response of the patient.

Supplied: In 15 cc. sterile dropper vials — 1%, 2%, and 4% strengths.

Contraindications: The use of Adsorbocarpine is contraindicated where pupiliary constriction is undesirable or hazardous such as in acute iritis. Miotics should be used with caution in hypertension.

Precautions: Avoid overdosage.

Adverse Reactions: There are no known side effects except slight ciliary spasm with resultant temporary reduction in visual acuity. Sensitivity is infrequently observed. Contact allergy or mucosal sensitization may develop after prolonged use. If signs of sensitivity develop during treatment, or if irritation persists or increases, the patient should be advised to discontinue use and consult the prescribing physician.

Caution: Federal (U.S.A.) law prohibits dispensing without prescription.

Tough cases
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Heavily pigmented, or brown, eyes are difficult to control with pilocarpine. Yet in Sherman's study with 26 heavily pigmented eyes, Adsorbocarpine® quickly brought IOP under control in these tough cases.

Following prolonged treatment with a pilocarpine/hydroxypropyl methylcellulose solution "20 of the 26 eyes (13 patients) were judged to exhibit uncontrolled IOP (above 21 mm Hg) . . . . In every instance, the eyes treated with the pilocarpine/Adsorbobase solution [Adsorbocarpine] were brought under control. In all but one patient . . . this reduction in pressure was accomplished at a greatly reduced pilocarpine concentration . . . .

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1. Sherman, Spencer E.: Clinical Comparison of Pilocarpi
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The widely accepted decongestant in Albalon® (naphazoline HCl) plus the antihistamine, antazoline phosphate, combine in Albalon-A to calm the itch that often accompanies allergic conjunctivitis and at the same time, decongest and whiten engorged ocular tissues.

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Further, Albalon-A is the only decongestant-antihistamine combination in the soothing artificial tear vehicle Liquifilm® (polyvinyl alcohol 1.4%). Less irritation. More comfort. Many more bouquets.

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Your next patients with blossoming allergic conjunctivitis will thank you.

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sterile ophthalmic solution

CONTAINS:
naphazoline HCl 0.05%, antazoline phosphate 0.5%, with Liquifilm (polyvinyl alcohol) 1.4%.

***INDICATIONS:** Based on a review of a related combination of drugs by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: For relief of ocular irritation and/or congestion or for the treatment of allergic, inflammatory, or infectious ocular conditions. Final classification of the less-than-effective indication requires further investigation.

CONTRAINDICATIONS: Hypersensitivity to one or more of the components of this preparation. **WARNING:** Do not use in presence of narrow-angle glaucoma. **PRECAUTIONS:** This preparation should be used only with caution in the presence of hypertension, cardiac irregularities or hyperglycemia (diabetes). **ADVERSE REACTIONS:** The following adverse reactions may occur: pupillary dilation, increase in intraocular pressure, systemic effects due to absorption (i.e., hypertension, cardiac irregularities, hyperglycemia).

ALLERGAN **Pharmaceuticals, Inc.**
Irvine, California 92713

AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 87 NUMBER 5 MAY, 1979

IMMUNOLOGIC CHARACTERIZATION OF OCULAR ADNEXAL LYMPHOID NEOPLASMS

DANIEL M. KNOWLES II, M.D., FREDERICK A. JAKOBIEC, M.D.,
AND
JAMES P. HALPER, M.D.
New York, New York

No group of ophthalmic tumors produces a higher percentage of diagnostic errors than the lymphoid neoplasms.[1,2] The ophthalmic pathologist usually makes secure diagnoses only with lesions that occupy the extreme poles of the histopathologic spectrum, that is, obviously benign inflammatory pseudotumors[3] and tumors composed of a monomorphous population of anaplastic and mitotically active lymphoid cells that almost certainly represent a malignant lymphoma.[4] Unfortunately, most lymphoid lesions lie between these extremes and diagnosis often depends on subtle, subjective judgments concerning the degree of cellular maturity.

The frequent inability to reliably distinguish benign from malignant lymphoid neoplasms is probably caused by reliance solely on histomorphologic criteria and failure to consider the immunologic heterogeneity of lymphoid cells, which cannot be detected cytologically. Current immunologic concepts (Table 1) separate human lymphocytes into two major populations: (1) T lymphocytes, which are thymus derived and principally responsible for cell mediated immunity; and (2) B lymphocytes, which are bone marrow derived, and responsible for antibody production. Investigational techniques for the demonstration of B- and T-cell markers have been widely applied to the study of neoplastic lymphoid cells isolated from peripheral blood and lymph nodal tissue,[5] resulting in three significant contributions to the understanding of systemic lymphoid malignancies.

First, these studies have generally determined the cell of origin of the systemic lymphoid malignancies. Second, clinicopathologic correlations with therapeutic and prognostic implications, not obvious or predictable by morphologic criteria alone, have been deduced. Third, and most important, systemic lymphoid malignancies are composed entirely of the progeny of a single neoplastic cell, that is, represent a monoclonal proliferation.[6,7] Benign reactive hyperplasias are polyclonal, containing the progeny derived from multiple cells. In malignant lymphoid infiltrates, B cells may bear more than one heavy-chain class of immunoglobulin, most commonly IgM and IgD, but they

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From the Department of Pathology (Dr. Knowles), the Algernon B. Reese Laboratory of Ophthalmic Pathology (Dr. Jakobiec), Edward S. Harkness Eye Institute, and the Department of Medicine (Dr. Halper), Columbia University, College of Physicians and Surgeons, New York, New York. This investigation was supported in part by grants EY02641 (Drs Knowles and Jakobiec) and CA24679 (Dr. Knowles) from the National Cancer Institute, and a grant from the Zelda Radow Weintraub Foundation.

Reprint requests to Daniel M. Knowles II, M.D., Department of Pathology, College of Physicians and Surgeons, Columbia University, 630 W. 168th St., New York, NY 10032.

TABLE 1

HUMAN MONONUCLEAR CELL MARKERS

Marker	Cell Distribution	Assay
Intrinsic surface membrane immunoglobulin	Defines B cells	Immunofluorescence Immunoperoxidase
Complement receptor	Most B cells; monocytes	Rosette assay
IgG Fc receptor	Most B cells; monocytes; some T cells	Immunofluorescence Rosette assay
Sheep erythrocyte rosette	Defines T cells	Rosette assay
Acid α-naphthyl acetate esterase	Monocytes, diffuse; T cells, focal	Histochemical
Surface antigens	B cells (Ia antigens)	Immunofluorescence Immunoperoxidase

bear only one class of light chain, either kappa or lambda.[5] Thus, monoclonal B-cell malignancies and polyclonal reactive lesions can be demonstrated on the basis of the similarity or dissimilarity, respectively, of the surface light-chain determinants of the constituent B cells.

Apart from a single case report,[8] the foregoing investigative techniques and biologic concepts have not been systematically applied to the study of ocular adnexal lymphoid neoplasms. To define more clearly and predict more accurately the biologic behavior of ocular adnexal lymphoid neoplasms, we correlated the light microscopic features of ten of these lesions with the results of selected immunologic and histochemical studies.

MATERIAL AND METHODS

The biopsy specimen from each of ten patients studied here was obtained fresh from the operating room and divided into portions suitable for routine histologic, electron microscopic, immunologic, and histochemical studies.

Mononuclear cell isolation—A portion of tissue was placed in tissue culture media RPMI 1640 supplemented with 10% fetal calf serum and teased until the cells were separated from the connective tissue stroma. The mononuclear cells were isolated by density gradient centrifugation.[9] Viability was determined to be 95% or greater by trypan blue exclusion.

Surface immunofluorescence and immunoperoxidase—Rabbit antihuman immunoglobulin antisera with specificities for γ, μ, α, δ, κ, and λ determinants were prepared as previously described elsewhere.[10,11] A heteroantisera directed against human Ia-like antigens, preferentially expressed on human B lymphocytes but not on T lymphocytes, was prepared by immunization of a rabbit with Ia alloantigen molecules isolated from human B lymphoblastoid cell lines.[12] Surface immunoglobulin and Ia antigens were demonstrated by either direct immunofluorescence[10] or direct immunoperoxidase.[13]

E_N rosette assay—Spontaneous rosette formation between isolated lymphoid cells and unsensitized sheep erythrocytes was assayed using *Vibrio cholerae* neuraminidase treated sheep erythrocytes.[14]

Acid α-naphthyl acetate esterase activity—Acid α-naphthyl acetate esterase activity as a marker for monocytes (diffuse cytoplasmic red-brown reaction product) and T lymphocytes (solitary red-brown reaction product) was demonstrated histochemically on cytocentrifuge smears of isolated lymphoid cells[15] and in cryostat sections prepared from tissue blocks fixed in buffered formal-sucrose.[16]

Microscopic examination of slides—The immunoperoxidase and acid α-naphthyl acetate esterase slide preparations were examined by conventional light microscopy. Immunofluorescent slide preparations were examined by a Leitz microscope equipped with alternating phase optics and incident fluorescent illumination with a filter system appropriate for fluorochrome stained preparations.

SUBJECTS AND METHODS

The ten patients included in this study represent unselected (Table 2) consecutive patients admitted or referred here, from whose lesions fresh tissue was made available for the performance of the investigative techniques previously outlined. The clinical course and features of four of these patients are detailed in the case reports. Nine of the lesions studied immunologically were predominately situated in the orbit (Figs. 1 and 2), and one orginated in the conjunctiva (Fig. 3).

CASE REPORTS

Case 7—A 57-year-old woman was referred here in March 1978 because of a firm, nontender nodule in the superonasal aspect of the left orbit behind the orbital septum. Results of physical examination were otherwise unremarkable. A chest x-ray film, bone scan, hemogram, bone marrow biopsy specimen, blood chemistry studies, and serum protein electrophoresis were normal. An excisional biopsy was performed and interpreted as a diffuse, poorly differentiated, lymphocytic lymphoma. The patient's medical history included a left lacrimal gland lymphoid infiltrate (biopsy performed elsewhere in 1975) and a subcutaneous nodule in the region of the left side of the forehead (biopsy also performed elsewhere in April 1977). Microscopic slides prepared from these earlier lesions were reviewed at the Armed Forces Institute of Pathology and also by us. They showed a diffuse lymphocytic lymphoma, cytologically similar to the present orbital biopsy material.

Case 8—A 59-year-old man was seen in March 1978 with a right lower eyelid mass (present for several months), right cervical adenopathy, and a mass in the right cheek. In 1967, he had undergone excision of a right cervical lymph node, interpreted then as a benign follicular hyperplasia. At the present time, ophthalmic examination revealed a freely movable, sausage-shaped mass 15 x 8 x 8 mm in size, lying horizontally at the right inferior orbital rim; the lesion did not involve the inferior fornix. Roentgenographic studies did not reveal orbital bone destruction. An excisional biopsy was performed and interpreted as an inflammatory pseudotumor. In May 1978, the patient underwent excisional biopsy of right buccinator and right cervical lymph nodes. We reviewed the cervical lymph node biopsy specimen, as well as the right cervical node biopsy specimen from 1967, originally interpreted as follicular hyperplasia; all of these specimens showed a nodular, poorly differentiated lymphocytic lymphoma.

Case 9—A 48-year-old man was referred here in September 1978, because of a salmon-colored subconjunctival mass, 15 x 12 x 5 mm in size. The results of physical examination were otherwise unremarkable. An excisional biopsy specimen revealed a diffuse, well-differentiated lymphocytic lymphoma shown to be a monoclonal B cell proliferation immunologically. Biopsy specimens of bilateral lower eyelid lymphoid lesions taken in 1970 in France, had been diagnosed as malignant lymphomas and treated by irradiation. A cervical lymph node biopsy specimen taken in 1974 and reviewed here showed a malignant lymphoma histologically similar to the present orbital lymphoma.

Case 10—A 49-year-old man initially came here in 1976 complaining of a progressive swelling of the left eye, present for two years. He had a marked left proptosis related to a large left orbital mass. A polyclonal gammopathy was discovered in the serum. An excisional biopsy specimen showed a plasmacytic reactive lymphocytic hyperplasia. In 1977 biopsy of a pulmonary nodule disclosed a plasma cell rich hyperplasia. The patient returned in 1978 with a recurrent mass over the left lower eyelid which bulged into the fornix with global displacement. Excisional biopsy again revealed a reactive lymphocytic hyperplasia with prominent reactive germinal centers and numerous plasma cells. This lesion was immunologically polyclonal, and the serum persisted to have a polyclonal gammopathy.

RESULTS

Light microscopy—Three distinctive histopathologic patterns were present in the ten patients: inflammatory pseudotumor (Fig. 4), reactive follicular hyperplasia (Figs. 5 and 6) and malignant lymphoma (Fig. 7).

TABLE 2

CLINICOPATHOLOGIC CHARACTERISTICS OF TEN OCULAR ADNEXAL LYMPHOID NEOPLASMS*

Patient No., Sex, Age, (yrs)	Symptoms	Ophthalmic Findings	History and Systemic Evaluation	Histopathologic Diagnosis of Present Orbital Biopsy	Therapy
1, F, 66	9 mo; right proptosis, pain, diplopia	Right proptosis, diplopia, right cranial nerve palsies II to VI, right afferent pupillary defect	Unremarkable	Inflammatory pseudotumor	Systemic corticosteroids
2, F, 63	12 mo; swelling of right lower eyelid and orbit	Mass, right inferior orbital rim	Unremarkable	Reactive lymphocytic hyperplasia	3,000 rads
3, F, 84	12 mo; enlarging mass, left superior orbit	Left proptosis, globe displaced downward, hard rubbery mass above left upper eyelid	Unremarkable	Lymphocytic lymphoma, poorly differentiated, diffuse	Surgery and 3,000 rads
4, M, 60	3 mo; fullness, right upper eyelid	Discrete, movable mass under right superior orbital rim	Unremarkable	Lymphocytic lymphoma, poorly differentiated, diffuse	Surgery and 3,000 rads
5, F, 63	5 mo; puffiness of left eye	Left proptosis, globe displaced down, forward, out	Unremarkable	Inflammatory pseudotumor	Surgery and 2,000 rads
6, M, 39	2 mo; enlarging painless mass, right lower eyelid	Firm, nontender, movable mass, fornix and right lower eyelid	Unremarkable	Reactive lymphocytic hyperplasia	Surgical excision
7, F, 57	Nontender mass, left orbit	Firm nodule, superonasal aspect left orbit	1975 left lacrimal gland, malignant lymphoma; 1977 forehead, malignant lymphoma	Lymphocytic lymphoma, poorly differentiated, diffuse	Surgical excision
8, M, 59	1 wk; inflammation of right eye	Palpable, movable mass, inferior aspect of right orbit	1967 cervical lymphoid hyperplasia (revised diagnosis: malignant lymphoma) 1978 cervical and buccinator lymph nodes, malignant lymphoma	Inflammatory pseudotumor	Surgical excision
9, M, 48	Swelling, right upper eyelid	Salmon colored subconjunctival mass extending into right upper eyelid	1970 bilateral lower eyelid lymphoid lesions. Diagnosis, lymphoma by history 1974 cervical lymph node, malignant lymphoma	Lymphocytic lymphoma, well differentiated, diffuse	3,000 rads
10, M, 49	Recurrent mass, left lower eyelid	Bulging mass, left lower eyelid displacement of globe, diplopia	1976 left orbital tumor, reactive lymphocytic hyperplasia 1977 pulmonary lesion, reactive lymphocytic hyperplasia	Reactive lymphocytic hyperplasia	3,000 rads

*These patients have been observed for from 9 to 15 months without further evidence of recurrent ocular adnexal or systemic lymphoid disease.

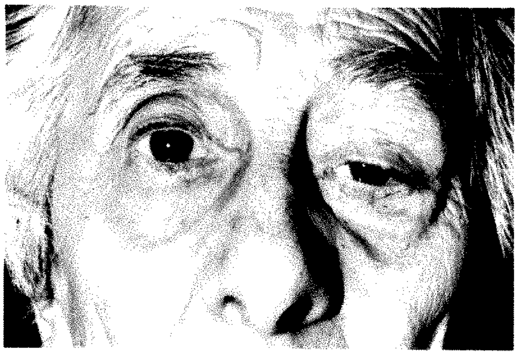

Fig. 1 (Knowles, Jakobiec, and Halper). An 84-year-old woman (Case 3) had painless left proptosis caused by a rubbery superior orbital mass.

Three of the ten patients (Cases 1, 5, and 8) had the histopathologic pattern of an inflammatory pseudotumor—a variably cellular infiltrate of small mature lymphocytes, bland histiocytes, and mature plasma cells superimposed on a matrix of alternately hyalinized and edmatous connective tissue. Reactive germinal centers were absent. The tissue was richly vascularized; endothelial lining cells were hypertrophic and extremely prominent.

Review of the nonorbital biopsy specimens of Case 8 revealed that both the earlier and concurrent lymph node biopsy specimens showed a nodular, poorly differentiated lymphocytic lymphoma. These lesions were discordant with the orbital biopsy specimen, which histologically and immunologically had the characteristics of an inflammatory pseudotumor (Fig. 8).

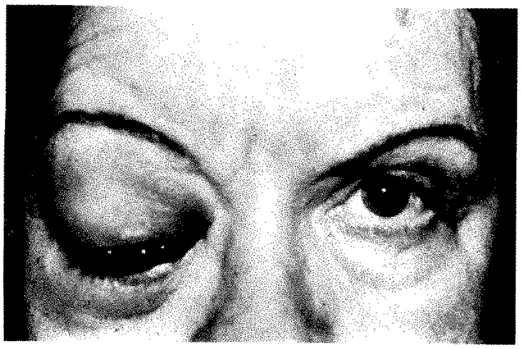

Fig. 2. (Knowles, Jakobiec, and Halper). A 66-year-old woman (Case 1) initially developed painful external ophthalmoplegia mimicking the Tolosa-Hunt syndrome, which evolved into an orbital inflammatory pseudotumor with proptosis and eyelid erythema.

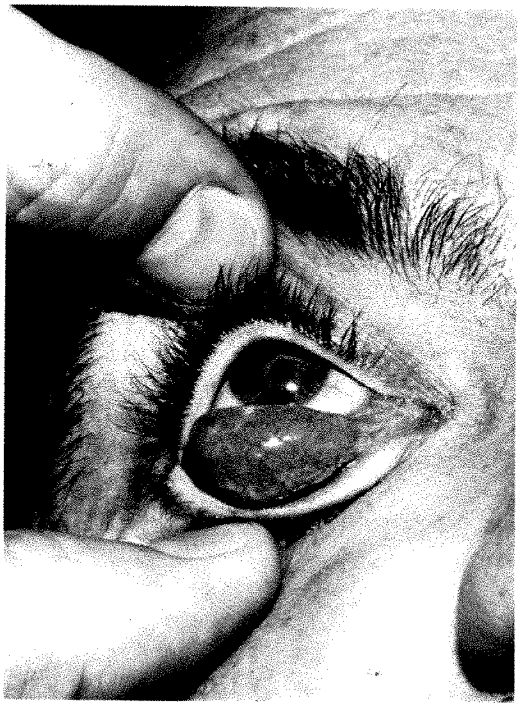

Fig. 3. (Knowles, Jakobiec, and Halper). A 39-year-old man (Case 6) developed a freely movable right inferior forniceal mass that could be prolapsed between the eyelids.

Three patients (Cases 2, 6, and 10) had the histopathologic pattern of reactive follicular hyperplasia—densely cellular infiltrates containing numerous reactive germinal centers. The interfollicular cells were small, fully mature, benign lymphocytes. Histiocytes and plasma cells, common in the inflammatory pseudotumors, were inconspicuous except in Case 4. These lesions were similarly well capillarized with prominent, hypertrophic endothelial lining cells.

Four patients (Cases 3, 4, 7, and 9) had lesions histologically consistent with a

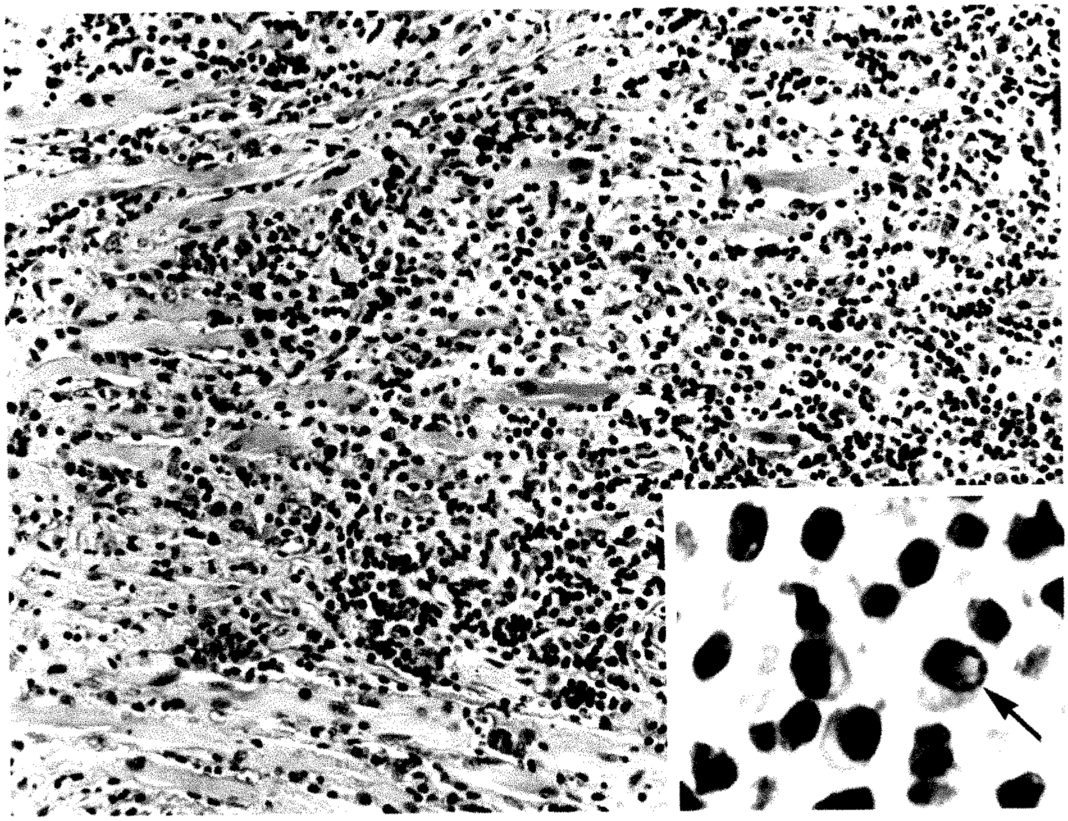

Fig. 4. (Knowles, Jakobiec, and Halper). Case 1. Inflammatory pseudotumor. A polymorphous, sparsely cellular infiltrate composed of small, mature lymphocytes, plasma cells, and bland reactive histiocytes diffusely infiltrate among skeletal muscle fibers. Inset, Note the mature chromatin pattern of the lymphocytes and the presence of plasma cells, and a Dutcher body (arrow), a PAS-positive intranuclear inclusion (hematoxylin and eosin, ×192; inset, ×1,100).

malignant lymphoma. Both the orbital biopsy specimen and the previous cervical lymph node biopsy specimen from Case 9 showed a diffuse proliferation of small, well-differentiated lymphocytes which occasionally contained nucleoli, consistent with a diffuse, well differentiated lymphocytic lymphoma. The lesions from two patients (Cases 3 and 4) consisted of a densely cellular lymphoid infiltrate in which an obviously immature, lymphocytic population predominated. These cells were larger than normal lymphocytes, possessed scanty cytoplasm, displayed occasional irregularly contoured (angulated) nuclei with clumped chromatin, and occasional nucleoli.

The biopsy specimen of Case 7 showed two histopathologic patterns (Fig. 9). One portion consisted mostly of small, mature-appearing lymphocytes, among which larger transformed lymphocytes, often with prominent nucleoli, were interspersed. Artifactual shrinkage during fixation and embedding permitted the latter cells to stand out, giving the lesion a starry sky appearance under low magnification. This fixation artifact precluded absolute certainty of the neoplastic nature of these cells. A contiguous portion consisted of a diffuse, monomorphous popu-

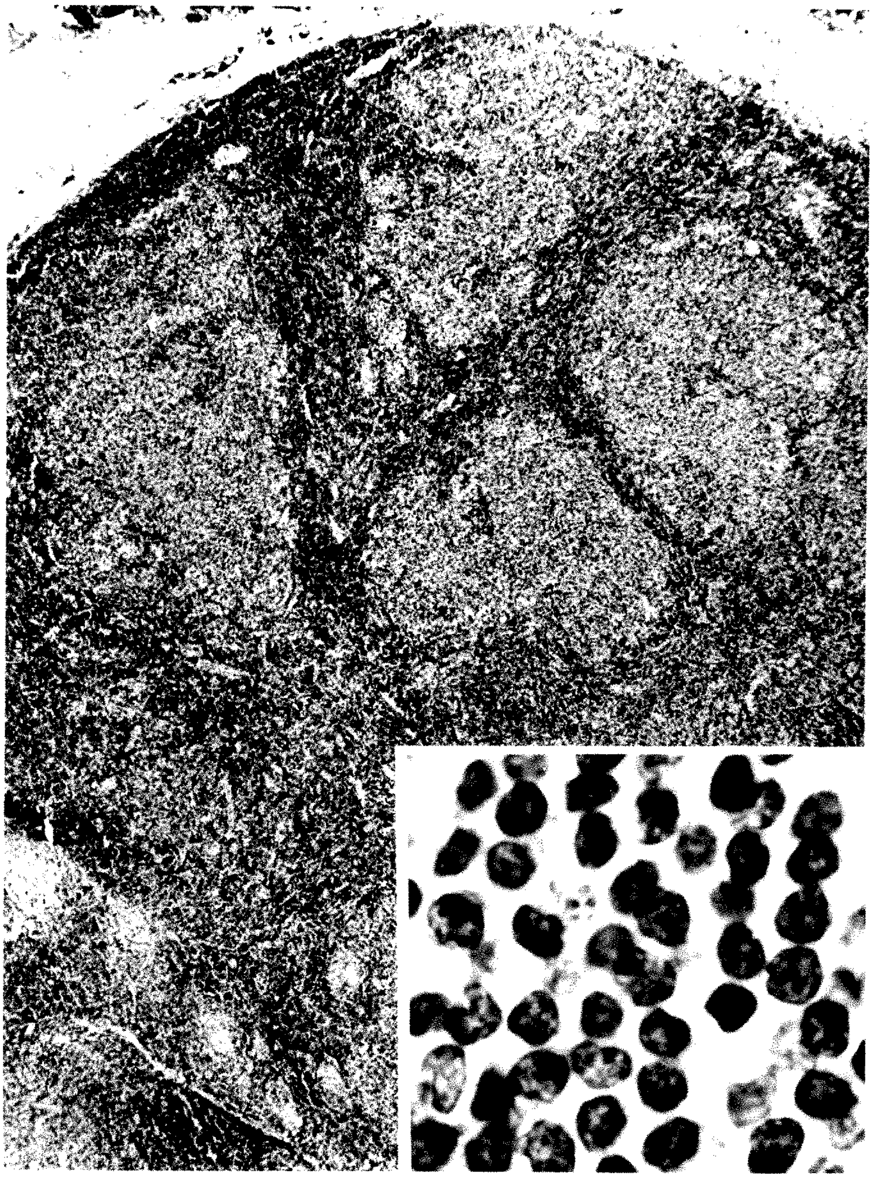

Fig. 5 (Knowles, Jakobiec, and Halper). Case 2. Densely cellular benign follicular hyperplasia. The hyperplastic infiltrate contains numerous reactive germinal centers. Inset, The interfollicular zones are composed of a uniform population of mature lymphocytes with a scarcity of other cell types. Despite histopathologic differences, this lesion and that in Figure 4 had similar polyclonal surface marker profiles (hematoxylin and eosin, ×38; inset, ×1,100).

lation of large, poorly differentiated neoplastic lymphoid cells with vesicular nuclei and prominent nucleoli, easily interpreted as a malignant lymphoma. The previous lacrimal gland and forehead lesions displayed sheets of poorly differentiated, neoplastic lymphocytes, cytologically similar to those seen in the present orbital biopsy specimen.

Cell surface marker studies—Im-

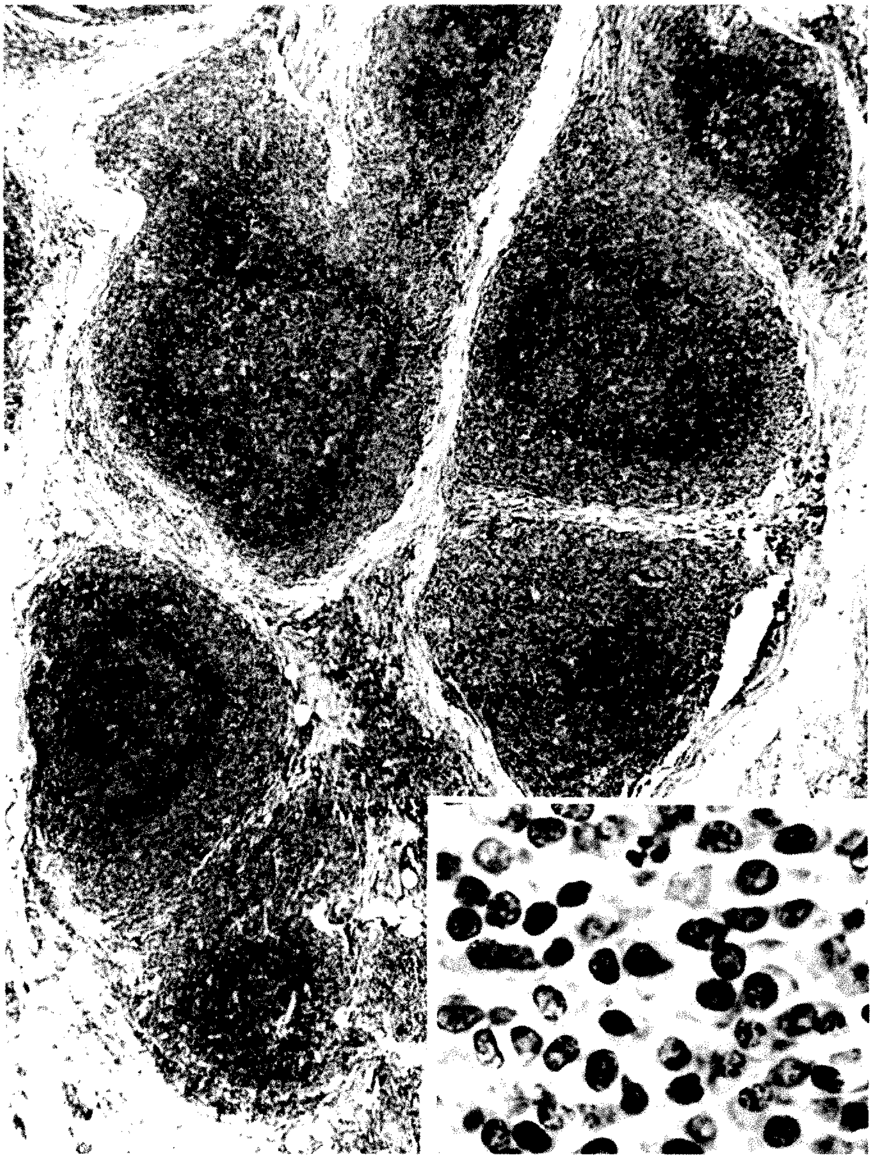

Fig. 6 (Knowles, Jakobiec, and Halper). Case 10. Benign reactive lymophoid hyperplasia. The lesion consists of numerous germinal centers separated by a densely collagenous stroma. Inset, Numerous plasma cells are present in the mantle zones of these germinal centers (hematoxylin and eosin, ×30; inset, ×600).

munologic characterization of the ten orbital lymphoid neoplasms, performed without reference to the light microscopic findings, resulted in the delineation of two clearcut patterns (Table 3). The lymphoid lesions were polyclonal in six patients and monoclonal in four patients. The six polyclonal lesions (Cases 1, 2, 5, 6, 8, and 10) had been interpreted histologically as inflammatory pseudotumors or reactive follicular hyperplasias. The four monoclonal lesions (Cases 3, 4, 7, and 9) had been interpreted histologically as malignant lymphomas.

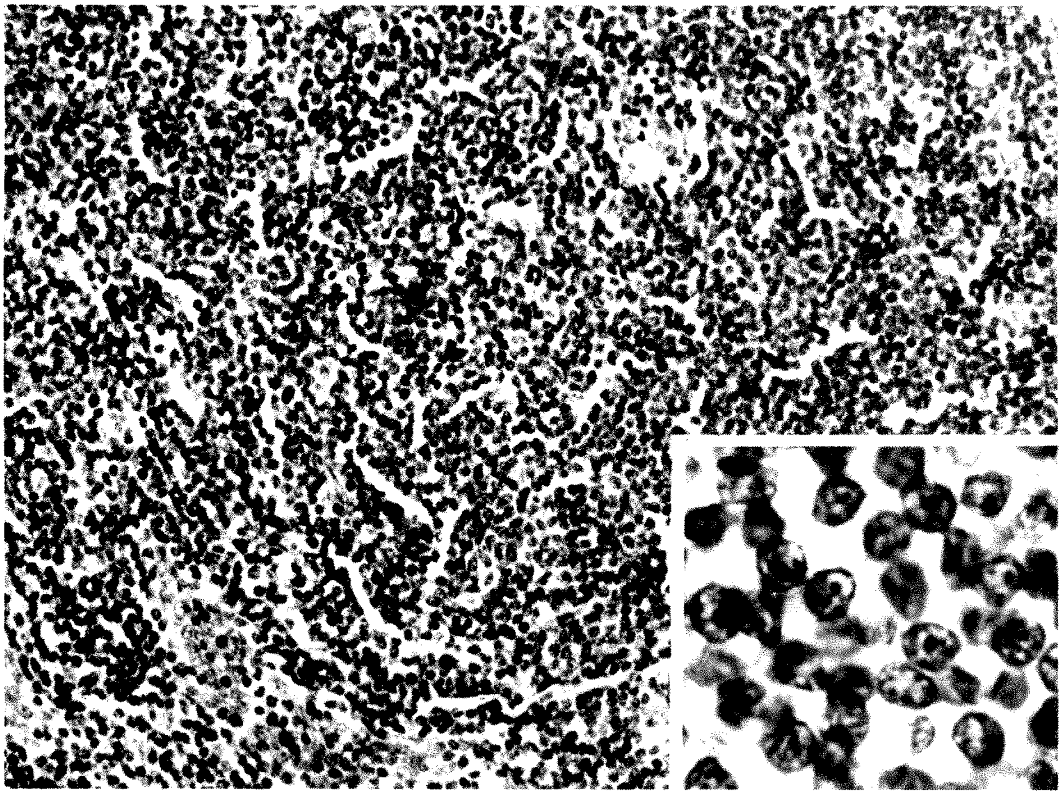

Fig. 7 (Knowles, Jakobiec, and Halper). Case 4. Diffuse malignant lymphoma. This featureless and densely cellular infiltrate is difficult to distinguish from benign lymphoid hyperplasia at low magnification. Inset, Higher magnification demonstrates the monomorphous neoplastic cytology: cells are larger than normal lymphocytes, possess thick nuclear membranes, clumped chromatin, and occasional nucleoli. Cell marker studies showed that this lesion represented a monoclonal B-cell proliferation (hematoxylin and eosin, ×208; inset, ×1,000).

The patients with a polyclonal orbital lymphoid infiltrate showed remarkably similar cell compositions as defined by cell surface markers, despite histopathologic diversity. In most instances, two thirds (47% to 73%) of the lymphoid (nonhistiocytic-monocytic) cells were T cells as determined by E rosette or focal acid α-naphthyl acetate esterase activity or both (Fig. 10). A rough quantitation of acid α-naphthyl acetate esterase positive cells (T cells) in cryostat sections confirmed these figures in those cases where the amount of biopsy tissue was sufficient to encompass this technique (Fig. 11). The remaining lymphoid cells in each case (27 to 60%) were B cells as determined by the presence of surface Ia antigens and immunoglobulins. In all six of these patients the constituent B cells were clearly polyclonal with respect to their surface light chain determinants, that is, in each instance B cells bearing surface kappa light chains and B cells bearing surface lambda light chains were present, kappa-bearing B cells predominating. These figures roughly approximate the normal polyclonal distribution of B and T lymphocytes in the normal and hyperplastic lymph nodes which we and other laboratories have studied.

Four patients (Cases 3, 4, 7, and 9) had a

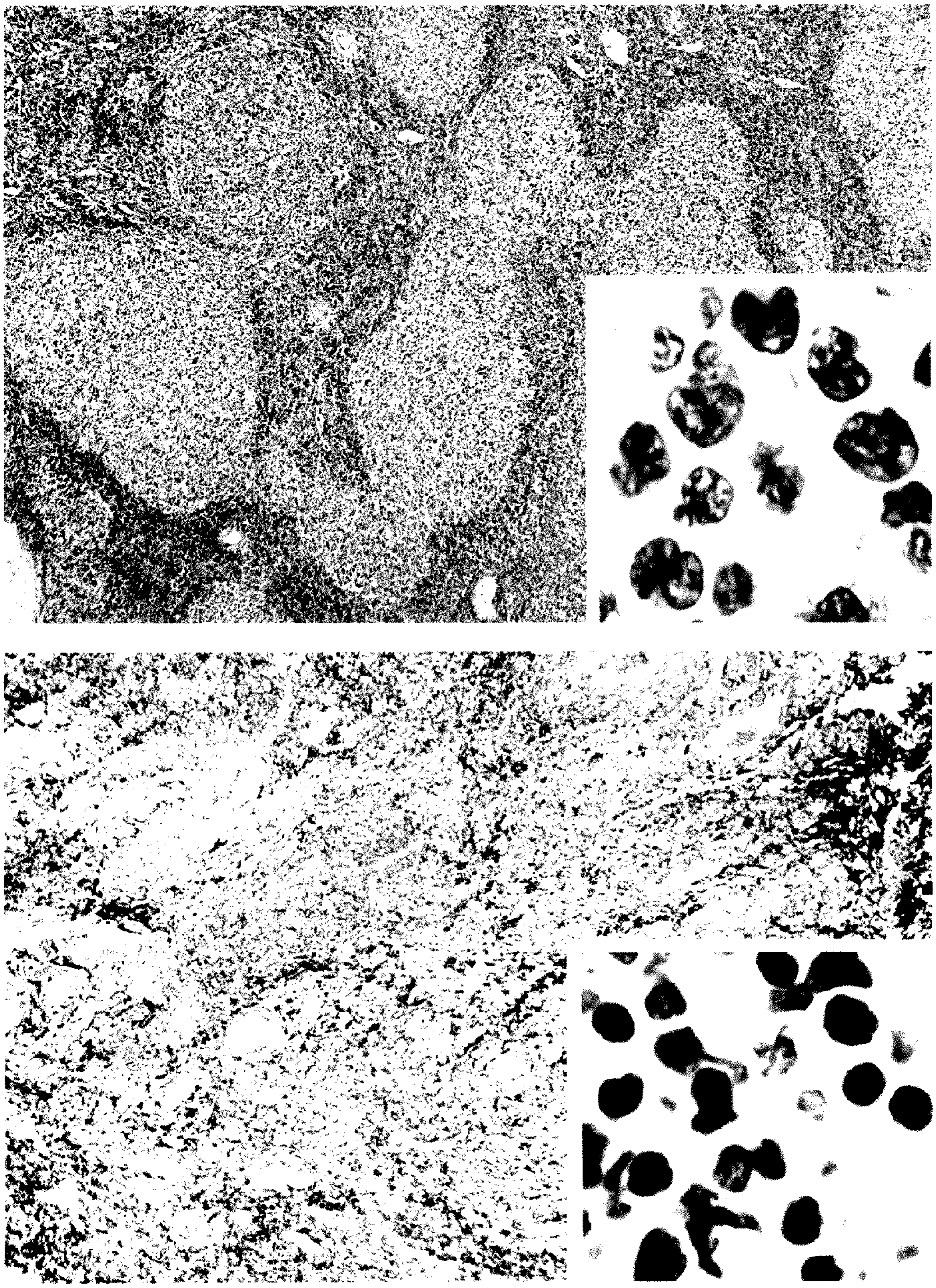

Fig. 8 (Knowles, Jakobiec, and Halper). Case 8. Top, Cervical lymph node biopsy. The lymph node architecture has been effaced by neoplastic lymphoid cells that form follicular centers; the cells lying inside and outside of these nodules are cytologically identical. Inset, The interfollicular neoplastic cells are larger than normal lymphocytes and have angulated, indented and cleaved nuclei. This is consistent with the poorly differentiated lymphocytic lymphoma, nodular type, in Rappaport's classification (hematoxylin and eosin, ×30; inset, ×1,000). Bottom, Orbital inflammatory pseudotumor, composed of a sparse and diffuse polymorphous infiltrate that exhibits considerable fibrosis. Inset, Higher magnification demonstrates the presence of a mixed population of benign, mature lymphocytes and histiocytes. Cell marker studies showed this orbital lesion to be immunologically polyclonal (hematoxylin and eosin, ×30; inset, ×1,000).

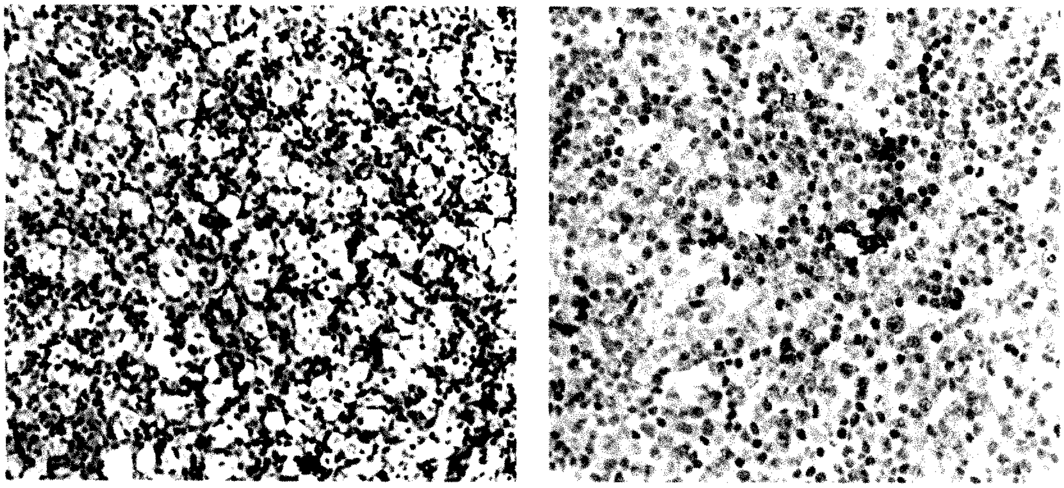

Fig. 9 (Knowles, Jakobiec, and Halper). Case 7. Orbital lymphoma. Left, One portion of the biopsy consisted mostly of small mature lymphocytes with interspersed larger cells that stand out because of shrinkage artifact, imparting a starry sky appearance. Right, Contiguous portion of the biopsy was composed of large, poorly differentiated neoplastic lymphocytes with scattered small mature lymphocytes. This zone was interpreted as a malignant lymphoma (hematoxylin and eosin; left, ×208; right, ×300).

monoclonal lymphoid infiltrate. One patient (Case 9) had 93% B cells, almost all of which bore IgMκ. Case 3 had 85% B cells, almost all of which bore surface IgMκ and IgDκ (Fig. 12). Case 4 had 83% B cells, the majority of which bore surface IgMκ. In each instance only a small percentage of T lymphocytes and B cells bearing surface light chain determinants of the other class were present. Case 7 had 71% B cells and 24% T cells; 80% of the B cells bore surface IgMκ immunoglobulin and 20% of them bore surface IgMλ immunoglobulin. These IgMλ bearing B lymphocytes and T lymphocytes may reflect the presence of a large polyclonal lymphoid focus in and around the monoclonal malignant lymphoma which is represented by the predominant IgMκ B cell population. This interpretation is consistent with studies in systemic lymphomas.

Discussion

The rate of error in the diagnosis of ocular and adnexal lymphoid tumors, that is, when the clinical outcome contradicts the histopathologic diagnosis, has been reported by Morgan[1-2] to be 20%. Recently, a review of approximately 60 borderline orbital lymphoid hyperplasias demonstrated almost a 50% mismatch in correlating pathologic diagnoses with clinical behavior.[17]

Careful application of the Rappaport[18] classification of systemic nodal lymphomas to extranodal lymphomas has improved the accuracy of diagnosis of ocular lymphoid neoplasms and the ability to predict clinical outcome.[19] However, 25% of lesions diagnosed as malignant lymphoma failed to disseminate.[19] Whether these lesions were simply misdiagnosed or were examples of successfully treated primary orbital lymphomas could not be determined in a retrospective light microscopic study.

There are drawbacks to the use of standard histomorphologic criteria in the evaluation of lymphoid infiltrates in extranodal sites such as the orbit. A preexistent lymph node architecture is absent and the usual diagnostic criteria for lymphoma, such as effacement of preexistent nodal architecture and capsular

TABLE 3

SURFACE MARKER CHARACTERISTICS OF TEN OCULAR ADNEXAL LYMPHOID NEOPLASMS*

	B Cell		T Cell	
Patient No.	Ia Antigens, %	SIg, %	E, %	ANAE, %
1	32	26 IgM, IgD 22 κ 5 λ	64	
2	27	17 IgM 19 κ 2 λ	68	
3	84	85 IgM, IgD 76 κ 4 λ	17	17
4	83	72 IgM 4 κ 75 λ		11
5	32	28 IgM 25 κ 6 λ		55
6		22 IgM, IgD 20 κ 8 λ		73
7	71	70 IgM 65 κ 9 λ	24	28
8	45	22 IgM 10 κ 2 λ	62	
9	88	88 IgM 93 κ 3 λ	3	3
10	50	60 IgM 26 κ 20 λ	47	

*Ia signifies surface antigens preferentially expressed on B lymphocytes, absent from T lymphocytes, detected by a heteroantisera; SIg signifies surface immunoglobulin. Cells were tested in each instance for all SIg determinants but only the determinants found to be present are tabulated. E signifies percentage of cells forming rosettes with neuraminidase treated, unsensitized sheep erythrocytes; ANAE signifies percentage of cells displaying focal acid α-naphthyl acetate esterase activity.

invasion, cannot be used. Consequently, the diagnosis depends entirely on the evaluation of cytologic detail. Increased experience with systemic nodal disease has shown that even extreme cytologic atypia, formerly thought to represent malignancy, can be present in benign reactive lymphocytic hyperplasias. Hyperimmune stimuli may bring about the blast transformation of small lymphocytes, resulting in atypical but still benign cells that can simulate lymphoma to an extraordinary degree.[20,21]

The use of modern immunologic and

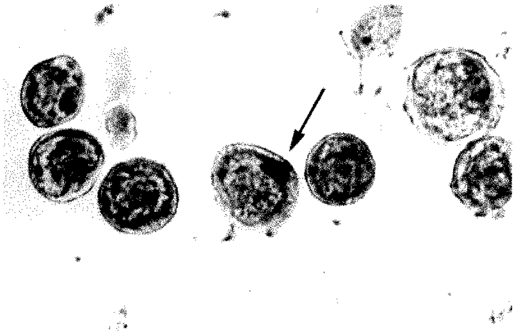

Fig. 10 (Knowles, Jakobiec, and Halper). Cytocentrifuge smear of cells isolated from the orbital biopsy from Case 7 and stained for acid α-naphthyl acetate esterase activity. The majority of the cells lacked such activity and bore surface Ia antigens (B cells). In this field there is only one T cell, demonstrated by the presence of a solitary nodule of red-brown reaction product (arrow). Note the nuclear detail (×1,400).

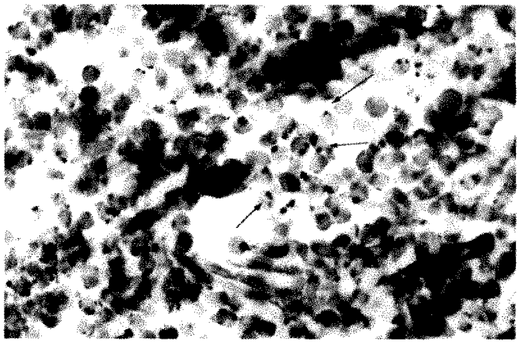

Fig. 11 (Knowles, Jakobiec, and Halper). Cryostat section prepared from a portion of the biopsy from Case 8 and stained for acid α-naphthyl acetate esterase activity. More than half of the cells display a focal dot of such activity (arrows) typical of T lymphocytes. This is consistent with the finding that 62% of the lymphoid cells were T cells as determined in cell suspension by E rosette assay (×600).

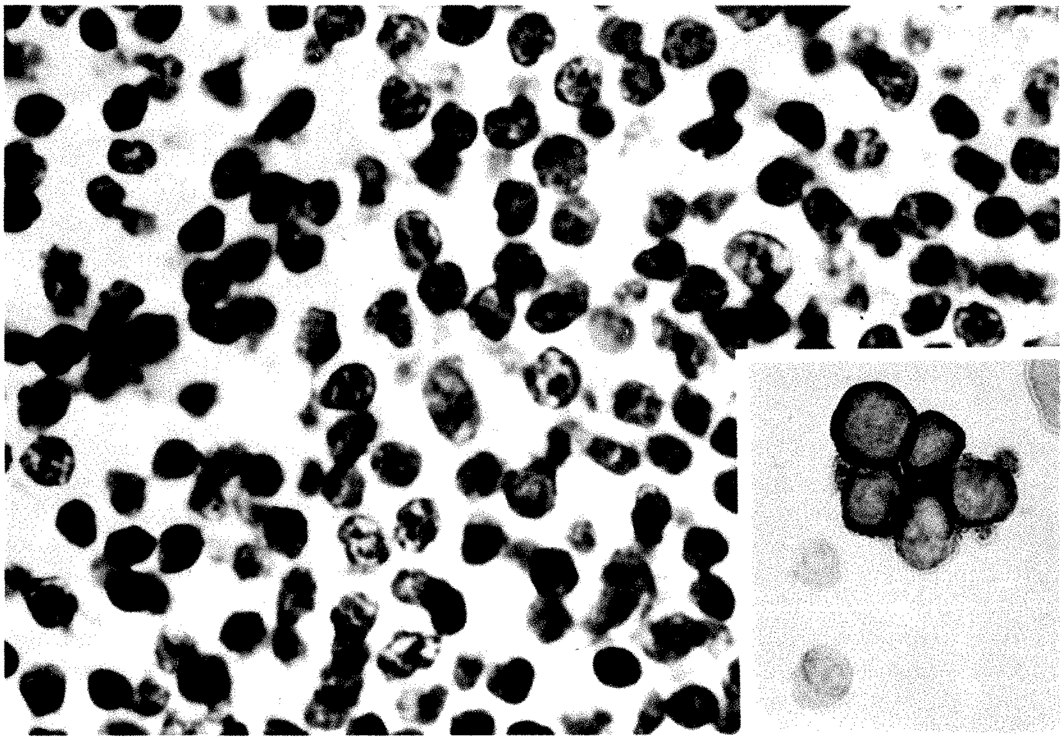

Fig. 12 (Knowles, Jakobiec, and Halper). Case 3. Malignant lymphoma. A patternless cellular infiltrate with neoplastic cytologic features similar to those in Figure 6. Inset, Immunoperoxidase staining demonstrated that the majority of the cells bore Ia antigens characteristic of B lymphocytes. These cells were monoclonal with respect to their surface light chain determinants (hematoxylin and eosin, ×1,100; inset, immunoperoxidase, ×600).

histochemical techniques which can identify cytologically identical but immunologically distinctive lymphocyte subpopulations holds promise for sorting out the diversity of ophthalmic lymphoid tumors. These methods have been applied to great advantage in the study of systemic nodal lymphoreticular disease,[5] but have been seldom used in the study of extranodal lymphoid neoplasms. The ocular adnexa may serve as a prototype in ascertaining the relevance to extranodal lesions of the results derived from the study of systemic lymphomas.

As anticipated from analyses of systemic lymphoid neoplasms, ocular adnexal lymphoid neoplasms are divisible, according to their surface marker characteristics, into two broad categories: polyclonal and monoclonal lesions. By using the histopathologic criteria for orbital lymphoid tumors previously outlined,[19] we classified the six polyclonal lesions as benign lymphoid pseudotumors or hyperplasias and the four monoclonal lymphoid proliferations as true malignant lymphomas, without reference to the results of the surface marker studies.

The lymphoid cell populations, as defined by cell surface markers, were strikingly similar in all six polyclonal cases, despite a spectrum of diverse, but benign, histomorphology. Inflammatory pseudotumors composed of a sparse, polymorphous infiltrate, hyperplasias consisting of a densely cellular and seemingly monomorphous infiltrate, and lesions with well developed reactive germinal centers all displayed a similar pattern of polyclonality, essentially identical to that observed in hyperplastic and reactive lymph nodes. Characteristically, about two thirds of the cells are T lymphocytes whereas one third are B lymphocytes, which are clearly polyclonal with respect to light chain determinants. In selected instances where reactive lesions are characterized by an abundance of germinal centers (B cell zones), B cells may predominate over T cells, as in Case 10. However, the B cells still remain polyclonal with respect to light chain determinants.

All of the lesions identified histopathologically as malignant lymphomas showed a strikingly similar pattern of B cell monoclonality, identical to that described in systemic nodal B cell neoplasms. Monoclonality was reflected in these cases by a predominance (in excess of 50%) of B cells bearing a single light chain class of surface immunoglobulin. Other admixed cell types (T cells and other B cells) may be reactive to the proliferating clone and are seen in most lymphomatous lymph nodes.[22]

Our basic hypothesis (benign reactive orbital lymphoid infiltrates are polyclonal, whereas malignant orbital lymphoid proliferations are monoclonal) seems plausible and worthy of continued evaluation. However, several key questions must be answered with respect to these findings, and certain reservations should be held with respect to generalizations based on these preliminary data.

The surface marker data must be correlated with the histopathology and interpreted within the clinical setting of each patient. Although our data, and especially that collected on systemic nodal lymphomas, support the concept that malignant lymphomas are monoclonal, a diagnosis of malignancy should not be based solely on cell surface marker studies. Indeed, in a monoclonal gammopathy the presence of a serum monoclonal protein spike can result from the proliferation of a single clone of immunoglobulin secreting plasma cells, which may or may not possess malignant potential.[23] Thus, both benign and malignant monoclonal gammopathies may exist. Similarly, a diagnosis of pseudotumor or pseudolymphoma should not be made solely on the findings of an immunologically polyclonal lymphoid population. The presence within a biopsy specimen of a small focus of monoclonal malignant lymphoma, surrounded by a large polyclonal lymphoid response, could yield a polyclonal pattern in marker studies. Case 7 had a somewhat less than straightforward monoclonal surface marker pattern, caused by the presence of a large focus of benign reactive lymphoid cells. Nonrepresentative sampling or the selective loss of one cell population in the preparation of a cell suspension could also yield inappropriate results.

Surface marker data are not an unequivocal prognosticator of the future development of systemic malignant lymphoma in a patient with an ocular lymphoid tumor. The demonstration of a monoclonal malignant lymphoma within the orbit does not necessarily imply the presence, or mandate the development of, generalized malignant lymphoma. Conversely, the demonstration of an immunologically polyclonal lymphoid pseudotumor of the orbit does not guarantee the absence of systemic lymphoma. For example, rarely, a patient may have two separate disease processes, a pseudotumor in the orbit and a systemic lymphoma. We have demonstrated the latter point clearly in this series (Case 8). Thus, monoclonality or polyclonality are not in themselves absolute prognostic indicators.

The systematic application of immunologic techniques to the study of orbital lymphoid neoplasms should provide insights into the histogenesis and biologic behavior of ocular adnexal lymphoid neoplasms which are not attainable by light microscopic observations. First, can an immunologically polyclonal benign orbital pseudolymphoma evolve into an immunologically monoclonal malignant orbital lymphoma? Some epidemiologic[24] and histologic evidence suggests that this transition may occur. The serial application of immunologic techniques to recurring ocular lymphoid lesions may resolve this question.

Second, what is the association between orbital malignant lymphoma and generalized lymphoma within the same patient? From earlier clinicopathologic studies[4,19] we learned that histiocytic lymphomas and poorly differentiated lymphocytic lymphomas of the orbit almost always represent an anomalous deposit of generalized lymphoma. This should be provable in any given case, because, *a priori*, all of the cells from the same malignant clone, regardless of the site of tissue involvement, will sport identical surface markers. This has been our experience with systemic lymphoid malignancies (unpublished data). Conversely, if each lymphomatous infiltrate represents coincidentally synchronous proliferations, then the cells may bear different constellations of surface markers. This has also been demonstrated in the case of systemic lymphomas.[25]

Third, can malignant lymphoma originate in the orbit and then disseminate systemically, and if so, what are the predominant patterns of spread? In our own extensive clinicopathologic review[19] of orbital lymphoid neoplasms, we were able to find only one patient who appeared to have a primary orbital lymphoma that disseminated to involve innumerable extranodal sites and eventually resulted in the patient's death. The use of surface markers should easily confirm whether such widespread systemic lesions represent manifestations of the original malignant clone originating in the orbit or a new malignant clone.

Fourth, are some orbital malignant lymphomas immunologically distinctive? We now know that the T cell lymphomas have a marked tendency to disseminate in an extranodal fashion, with a special proclivity to involve the skin and central nervous system.[26] One might expect then that many lymphomas both originating in, and secondarily involving, the orbit might be of T-cell origin. Although all four malignant lymphomas in the present series were of B-cell origin, the series was too small to provide sufficient data.

SUMMARY

We correlated the light microscopic features of ten ocular adnexal lymphoid neoplasms with the results of selected immunologic and histochemical lymphocyte marker studies. The lesions were divided into three histomorphologic patterns: inflammatory pseudotumor, reactive follicular hyperplasia, and malignant lymphoma. The six inflammatory pseudotumors and reactive follicular hyperplasias, despite diverse histomorphology, were immunologically polyclonal and consisted of from 45 to 73% T cells and 27 to 60% B cells, the B cells being polyclonal with respect to light chain determinants. The four malignant lymphomas were immunologically monoclonal. Each consisted of a predominant B-cell proliferation, greater than 50% of the cells, which were monoclonal with respect to surface light chain determinants. No T-cell proliferations were observed. Our preliminary data indicate that, as in systemic lymphoid neoplasms, benign reactive lymphoid hyperplasias are immunologically polyclonal whereas malignant lymphomas are immunologically monoclonal. The systematic application of immunologic and histochemical techniques to the study of ocular adnexal lymphoid neoplasms may elucidate the natural history of these lesions.

ACKNOWLEDGMENTS

Chang Yi Wang and Robert Winchester, M.D., provided the Ia heteroantisera. Karl Perzin, M.D., reviewed the histologic preparations.

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OPHTHALMIC MINIATURE

Thorndyke laughed softly. "The human field of vision, Anstey," said he, "as measured by the perimeter, is well over a hundred and eighty degrees. It doesn't take much lateral movement of the head to convert it into three hundred and sixty. The really important factor is not optical but mental."

R. Austin Freeman, *The Cat's Eye*
New York, A. L. Burt Co., 1927

WHAT PATIENTS RECALL OF THE PREOPERATIVE DISCUSSION AFTER RETINAL DETACHMENT SURGERY

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The concept of informed consent that encompasses both physician liability and patient's rights is not new. In 1914, Justice Benjamin Cardozo stated, "Every human being of adult years and sound mind has a right to determine what shall be done with his own body. . . . " It is widely accepted that the purpose of informed consent before surgery is to provide adequate information to the patient so that he or she can make an intelligent decision about a given medical or surgical procedure. Such information should include diagnosis, modes of therapy, reasonable alternatives, risks, and prognosis. Important to the preoperative discussion is the degree of actual patient understanding and retention. We report herein the results of a prospective study conducted to determine to what extent preoperative information, informed consent, is retained by the patient during the immediate postoperative period after a scleral buckling procedure.

SUBJECTS AND METHODS

One hundred patients undergoing a primary scleral buckling procedure at Mayo Clinic for rhegmatogenous retinal detachment were included in this study. Excluded from this consecutive series were mentally handicapped patients, those under the age of 18 years, or patients who had retinal surgery during the previous 12 months. During a nine-month period, 63 men and 37 women were included in this study. Age distribution was comparable to other studies of retinal detachment patients[2,3] (Fig. 1).

Before surgery each patient was given a detailed 15 to 20 minute discussion by one of us. All preoperative discussions were given in simple medical and lay terms. Diagnosis, modes of therapy, risks, reasonable alternatives, prognosis, and visual rehabilitation were discussed by using a standard format. A check-off list was used by us to validate that our preoperative disclosure was simple and uniform for all patients. A model of the eye was used for illustrative purposes during this explanation. In addition to the thorough preoperative disclosure, the patients were asked if they had questions concerning any aspects of these discussions. Every possible attempt was made by us to completely answer all the patients' questions. Patients were unaware that they would be involved in a study during the preoperative discussions. During the postoperative hospitalization, each patient, after consenting to participate in this study, was asked 12 standardized questions regarding their ocular disorder (Table). The interval between the preop-

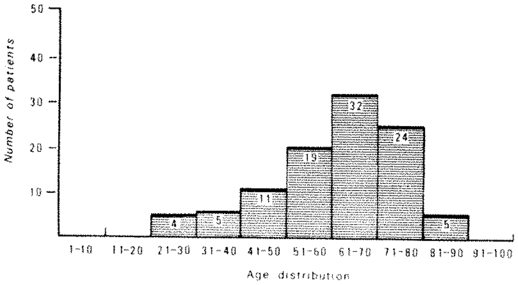

Fig. 1 (Priluck, Robertson, and Buettner). Age distribution in years of 100 patients treated with scleral buckling procedure for rhegmatogenous retinal detachments.

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TABLE

LIST OF QUESTIONS DIRECTED TO THE PATIENTS DURING IMMEDIATE POSTOPERATIVE PERIOD

1. Do you think you had an adequate explanation given to you by the (your) physician preoperatively; and were all of your questions answered?
2. Why did you have eye surgery?
3. What caused this condition?
4. What would most likely have happened if you did not have surgery?
5. What risks are involved in this type of operation?
6. Which one of the following methods was used?
   a. Laser beam
   b. Freezing
7. Approximately what percent of success is there in this particular operation?
8. Does successful reattachment of the retina mean your vision is likely to fully recover?
9. What alternatives to surgical treatment did you have?
10. How many weeks after surgery will you be able to return to your full activities?
11. Was there any foreign material (plastic or sponge) purposefully left around your eye after surgery?
12. When will you receive a prescription for new glasses?

erative informed consent and questionnaire was two to 11 days with a mean of 4.2 days. Every possible effort was made to be liberal in determining whether a question was adequately answered. An inadequate response included either a wrong answer or an "I don't know" response. If a question was inadequately answered, the patient was then asked if he or she remembered this particular subject being discussed during the preoperative explanation.

RESULTS

Ninety seven percent of the patients stated that they had a satisfactory preoperative discussion and had all of their questions adequately answered. However, the overall mean accuracy for patient retention in the immediate postoperative period for all 12 questions was only 57% (Fig. 2). Questions that related to similar concepts were then grouped into five categories: (I) diagnosis and the nature of illness (questions 2 and 3); (II) natural history and reasonable alternatives (questions 4 and 9); (III) surgical technique and expected result (questions 6, 7, 8, and 11); (IV) visual rehabilitation (questions 10 and 12); (V) surgical risk (question 5). The percentage of accurate patient retention for these five categories is summarized in Figure 3.

Whereas 97% of the patients believed they had a satisfactory preoperative discussion, 54% of the time, patients who gave inadequate responses denied the subject had ever been discussed preoperatively (Fig. 4). Such denials were particularly evident for question 3, relating to retinal detachment being caused by a retinal hole (62 of 79 [78%] denied), question 5 concerning surgical risk (58 of 77

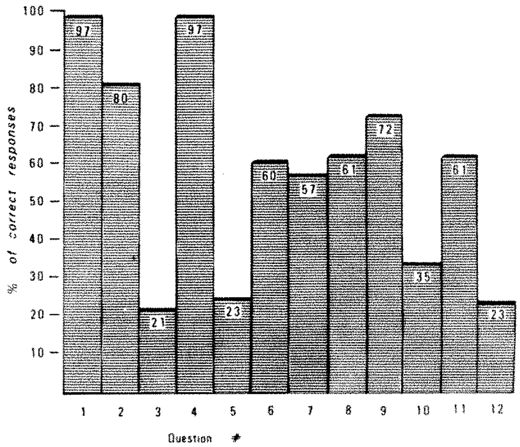

Fig. 2 (Priluck, Robertson, and Buettner). Percentage of correct responses for each question asked during the immediate postoperative period.

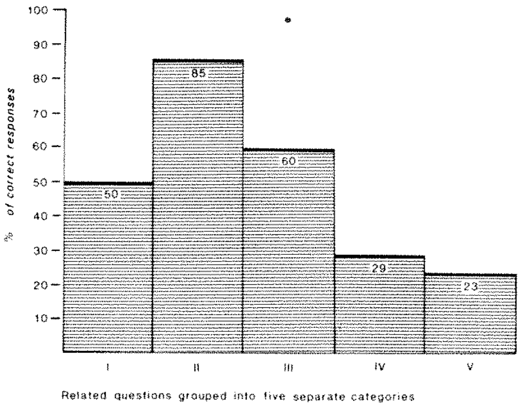

Fig. 3 (Priluck, Robertson, and Buettner). Percentage of patients providing correct responses for each of five categories. (I, diagnosis and nature of illness (questions 2 and 3); II, natural history and reasonable alternatives (questions 4 and 9); III surgical technique and expected result (questions 6, 7, 8, and 11); IV, visual rehabilitation (questions 10 and 12); and V, surgical risk (question 5).

[75%] denied), question 9 relating to alternative methods (24 of 28 [86%] denied), and question 11 regarding the placement of a permanent scleral buckling element (31 of 39 [79%] denied).

DISCUSSION

The doctrine of informed consent has recently gained attention as patients seeking medical assistance fail to achieve their preconceived expectations. Pertinent to informed consent is the patient's understanding of the preoperative disclosure. However, actual patient retention of the preoperative information has generally received little attention.

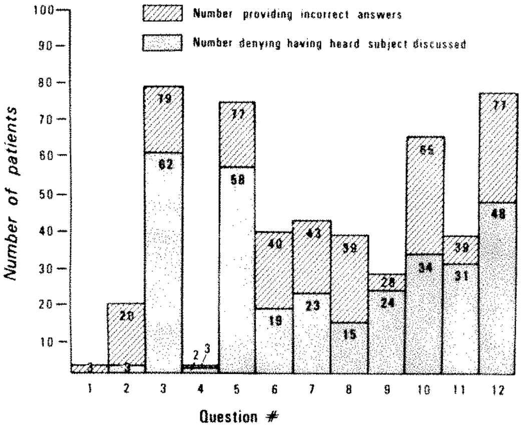

Fig. 4 (Priluck, Robertson, and Buettner). Number of patients providing incorrect answers to each question (identified by the total height of each column) and the number of these patients who denied hearing the subject discussed (identified by the height of the solid-lined column).

Robinson and Merav[4] have shown that overall patient retention of preoperative information was 29% for primary recall during a four- to six-month postoperative period after open heart surgery. Leeb, Bowers, and Lynch[5] reported the retention rate of 35% for 100 consecutive preoperative patients and emergency room patients. Overall patient retention of preoperative disclosure in this study was 57%.

Even though the percentage of patient retention after surgery was significantly higher in our study as compared to previous investigations, it is still low. It should be emphasized that this study was conducted in the early postoperative period with a mean interval of 4.2 days between preoperative disclosure and the postoperative questionnaire. Several months or years after surgery, retention rates would be expected to decrease.

Although patients acknowledged a thorough preoperative discussion (97%), and were aware of the consequences of the probable natural course of retinal detachment (blindness), with 97% accuracy, the patients generally retained little regarding specific information given during the preoperative explanation. They retained minimal knowledge of the involved surgical risk. Only 23% of patients recalled that hemorrhage or infection could possibly occur intraoperatively or postoperatively, and only 3% were aware that either of these complications could essentially destroy the eye. Sixty-one percent of patients did not remember that a foreign material was permanently sutured to the eye. If a patient inadequately answered a question, more than one-half of the time there was denial that information relevant to the question was ever discussed.

Preoperative disclosure is of paramount importance in supporting the validity of the informed consent doctrine. It is disturbing to learn that overall patient recall is far from optimal even when special efforts by the physician are made to provide an understandable and thorough preoperative disclosure. Additionally, a wide variation exists in what the patient remembers of the various aspects of informed consent: diagnosis, alternatives, risks, prognosis, contemplated procedures, and visual rehabilitation. Patient failure to accurately recall preoperative discussions undermines the concept of informed consent to safeguard his own self-determination.

Why does the patient fail to recall preoperative disclosure accurately? Anxiety, apprehension, and suffering may all contribute to the disruption of an individual's normal cognitive faculties. Further obscuration of preoperative disclosure may be caused by the patient's preconceived notion of the surgery and expected results, unconscious suppression of unpleasant information, or perhaps a lack of alertness. Preoperative explanations using complicated scientific terminology should be avoided. Despite the use of simple medical and lay terms, the percentage of patient retention will probably be low as shown in our study.

The results of this study indicate that patients retain primarily those points of the preoperative disclosure that seem to be in favor of their decision to have surgery. They are likely to suppress what makes them anxious or may make the therapy seem threatening. This emphasizes the importance of establishing a relationship of trust between doctor and patient so that the patient can make a decision to undergo specific therapy with confidence, even though he or she may not remember details of the preoperative disclosure.

SUMMARY

We conducted a prospective study to determine the percentage of preoperative information that was retained by 100 patients undergoing a scleral buckling procedure. Each patient received a standardized preoperative discussion regarding the nature of retinal detachment and its management. During the immediate postoperative period, each patient was questioned about the informed consent discussion. Although 97% of the patients acknowledged a thorough preoperative discussion there was a definite disparity between the patients' concept of adequate physician disclosure and the actual retention of the preoperative explanation. Overall retention of preoperative disclosure based on correct answers to 12 questions was only 57%. Few patients, 23%, retained knowledge of surgical risks, and only 61% remembered that a foreign material would be permanently sutured to the eye. When a patient failed to remember the correct answer to a question, more than half of the time he or she denied that information relevant to the question had been discussed. The results of this study indicate that patients retain primarily information that seems to be in favor of their decision to have surgery.

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SECOND TUMORS IN NONIRRADIATED BILATERAL RETINOBLASTOMA

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Bilateral retinoblastoma occurs in approximately 30% of all cases of retinoblastoma in this country.[1] Patients with bilateral retinoblastoma usually have significant asymmetry of the disease, and the eye diagnosed as having retinoblastoma is more involved. The more involved eye is treated with enucleation, whereas the second eye is treated with external beam radiation, cobalt plaques, xenon arc photocoagulation, or cryopexy.[2] It is unusual to treat bilateral retinoblastoma initially with bilateral simultaneous enucleation unless both eyes are extensively involved with tumor, there is no metastatic disease, and no useful vision could be expected with less radical treatment.[1]

We reviewed all bilateral retinoblastoma cases seen here between 1922 and 1978 and report herein a review of cases of those patients treated with bilateral enucleation alone and without irradiation. We determined survival in patients treated with bilateral enucleation without any other forms of treatment and explored the incidence of second nonocular tumors in these patients.

MATERIAL AND METHODS

Retrospective chart reviews were made of all cases of retinoblastoma on file here between 1922 and 1978; 1,323 cases were reviewed. Patients selected for this series were patients treated with bilateral enucleation alone. Many of the cases had bilateral simultaneous enucleation, although in others, the second enucleation was delayed. If either eye received irradiation or any other additional form of treatment, that patient was excluded from this series. Cases in which recurrence of tumor led to subsequent irradiation of the orbit after bilateral enucleation were also excluded from this series. Follow-up clinical data were obtained by letters, office records, hospital charts, and the like, and the histopathology on all cases of second tumors was reviewed. Two of these cases were reviewed elsewhere and were included in another series.[3]

RESULTS

Of the 1,323 cases reviewed, 36 patients with bilateral retinoblastoma were initially treated solely with bilateral enucleation. Eight of these patients received postoperative irradiation and were excluded from this series; 16 patients had simultaneous bilateral enucleations; and 12 had enucleation separated by a time interval of two weeks to 14 months, with a mean of 4.7 months.

Four of the 28 patients in this study were lost to follow-up immediately after their enucleations. Follow-up on the remaining 24 has ranged from six months to 32 years. Metastatic retinoblastoma occurred in two of these 24 patients and was fatal in each case. An autopsy was performed that confirmed death caused by metastatic retinoblastoma. The survival rate for advanced bilateral retinoblastoma treated with bilateral enucleation alone was 92% (22 patients). Of the 22 survivors of bilateral retinoblastoma, three (14%) developed second tumors. The average latent period after enucleation for the development of second tumors was 15.3 years with a range of ten to 23 years. The

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age at diagnosis of bilateral retinoblastoma ranged from two months to 36 months with a mean of 14.5 months.

CASE REPORTS

Case 1—An 11-month-old boy had bilateral retinoblastoma. Both eyes were Group 5 Reese-Ellsworth classification and were simultaneously enucleated. Pathologic examination of the specimens revealed retinoblastoma without any evidence of extraocular extension. He did well until 13 years later when a cystic mass was found in the left temporal muscle. Biopsy revealed undifferentiated rhabdomyosarcoma (Fig. 1). Despite treatment of this second tumor, the patient developed pulmonary metastasis and died.

Case 2—A 9-month-old girl was seen with advanced Group 5 bilateral retinoblastoma. Tests for metastasis were negative. She underwent simultaneous bilateral enucleation and did well until ten years later when pain in the right leg occurred and she developed a limp. A biopsy revealed osteogenic sarcoma of the right femur (Fig. 2). Despite treatment, she developed pulmonary metastasis and died.

Case 3—An 8-month-old girl with bilateral retinoblastoma, advanced Group 5, underwent simultaneous enucleation. She did well until 23 years later, when a melanotic lesion of the left thigh was noted and excised. The pathologic diagnosis was malignant melanoma (Fig. 3). She developed regional node metastasis and was given chemotherapy. One year later she was hospitalized for pulmonary pelvic and possibly hepatic and bone metastasis from the original melanoma. The patient died of widespread metastatic disease.

DISCUSSION

The survival rate (92%) in our series is comparable to the survival rate of patients treated with enucleation and irradiation.[1] It seems that bilateral enucleation is justified in those cases where there is no evidence of metastatic disease and both eyes are extensively involved.

The association of retinoblastoma with second nonocular neoplasms has been commented on by many but only recently has it been well understood. Reese, Merriam, and Martin,[4] in the first large series of patients with bilateral retinoblastoma treated with radiation and surgery, reported two cases of second tumors in 55 retinoblastoma patients. The rela-

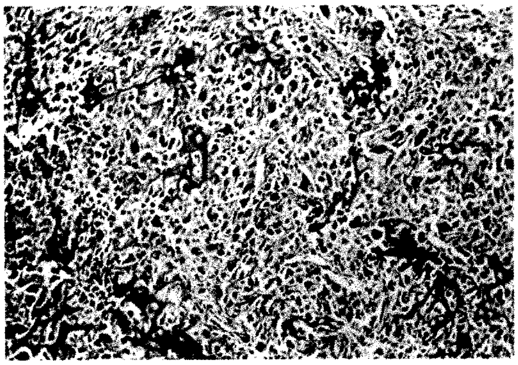

Fig. 2 (Abramson, Ronner, and Ellsworth). Case 2. Osteogenic sarcoma of the right leg ten years after bilateral simultaneous enucleation for bilateral retinoblastoma.

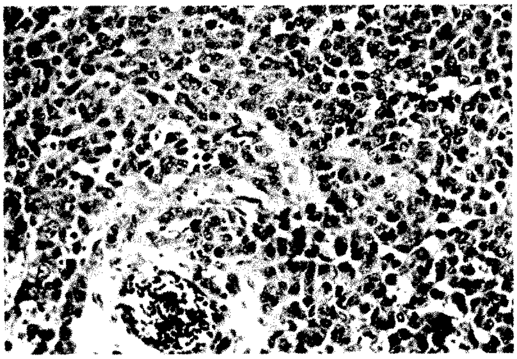

Fig. 1 (Abramson, Ronner, and Ellsworth). Case 1. Undifferentiated embryonal rhabdomyosarcoma of left temple in patient 13 years after bilateral simultaneous enucleation for bilateral retinoblastoma.

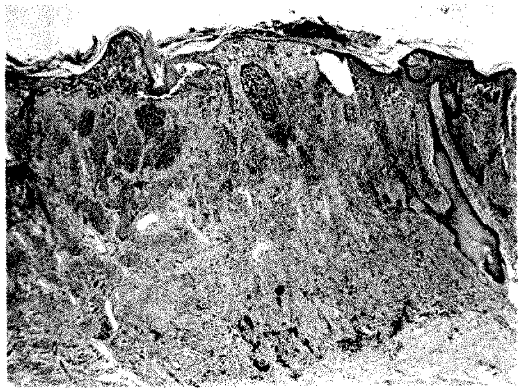

Fig. 3 (Abramson, Ronner, and Ellsworth). Case 3. Metastatic cutaneous malignant melanoma 23 years after bilateral simultaneous enucleation for bilateral retinoblastoma.

tionship between radiation therapy and second malignancies was emphasized by Forrest[5] who reported second nonocular tumors after successful radiation treatment for retinoblastoma. It was his impression that these tumors were secondary to radiation because the tumors appeared: (1) in the field of radiation treatment, (2) after long latent periods, and (3) after high doses of radiation. This concept that radiation induced neoplasms in patients treated with retinoblastoma has been supported by others.[6,7] The occurrence of these tumors has been reported by others,[8-11] and many investigators continue to implicate the radiotherapy for the development of these second nonocular tumors. In a study of over 2,300 cases from the Columbia Presbyterian Medical Center and the Armed Forces Institute of Pathology,[3] we reported that the retinoblastoma patients who develop second tumors had originally had bilateral retinoblastoma. At the Armed Forces Institute of Pathology, only 19% of 1,200 patients with retinoblastoma had been treated for bilateral retinoblastoma. Despite this, 98% of the patients from the Armed Forces Institute of Pathology who developed second tumors had been treated for bilateral retinoblastoma. When both the Columbia Presbyterian and Armed Forces Institute of Pathology were combined, 78 of the 80 patients who developed second nonocular tumors had originally been treated for bilateral retinoblastoma. Of all survivors of bilateral retinoblastoma from the Armed Forces Institute of Pathology, 12.6% had developed a second tumor, and of all survivors from Columbia-Presbyterian Medical Center, 10% had developed a second tumor.

All patients with bilateral retinoblastoma harbor the germinal mutation that is passed on as an autosomal dominant. The tendency for the development of these second tumors is linked to the germinal mutation of retinoblastoma and not necessarily to the treatment with radiation. Additional evidence for this theory was presented in the combined Columbia-Presbyterian Medical Center and the Armed Forces Institute of Pathology series. Fifteen patients in this series developed tumors out of the field of radiation, and it was emphasized that retinoblastoma patients had an extremely high incidence of nonocular tumors distant from the radiation site.

In our present series, one patient developed an undifferentiated embryonal rhabdomyosarcoma of the temple that would have been called a radiation induced tumor, because it followed a long latent period and was in the area of the radiation beam, except that the patient had been treated only with bilateral enucleation. The most common distant nonocular tumor seen in survivors of bilateral retinoblastoma is an osteogenic sarcoma,[3] and one patient in our series developed an osteogenic sarcoma.

The incidence of second neoplasms in patients who survive treatment of bilateral retinoblastoma is similar whether the patients received radiation or not. Because of the long latent period involved in many of these tumors, the incidence will probably be between 15 and 20% of all patients with bilateral retinoblastoma who survived retinoblastoma treatment. The role of radiation in inducing any of these tumors is not certain, and the patient with the germinal mutation for retinoblastoma is more at risk for the development of second nonocular tumors whether they receive radiation or not. The death rate from second nonocular tumors may be higher than the death rate from bilateral retinoblastoma, regardless of treatment.

SUMMARY

We reviewed the records of 1,323 patients with retinoblastoma treated here between 1922 and 1978. Twenty-eight patients underwent bilateral enucleation alone and received no additional treatment for their advanced tumors. Of the 28

patients, 22 (92%) survived. Four patients were lost to follow-up, and two died of metastatic retinoblastoma. Of 22 survivors of bilateral retinoblastoma treated with enucleation alone, three developed second tumors at a mean of 15.3 years after bilateral enucleation. One patient developed a rhabdomyosarcoma in the left temple region, the second patient developed an osteogenic sarcoma of the right femur, and the third developed a malignant melanoma of the left thigh. All three patients died of their disease. The incidence of second tumors in patients who survived bilateral retinoblastoma treated without radiation was 14% (three patients), which is comparable to a series previously reported in which patients were treated with enucleation and irradiation.

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RETINAL DETACHMENT AFTER PHACOEMULSIFICATION

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The incidence of retinal detachment after phacoemulsification with posterior capsule surgery appears to be about the same as that after intracapsular cataract extraction.[1] We report herein the clinical characteristics of retinal detachments after phacoemulsification and our surgical results dealing with this complication.

MATERIAL AND METHODS

We examined 102 eyes of 89 patients with retinal detachments after phacoemulsification was performed. In two of these patients successful retinal surgery had been performed elsewhere several months before redetachments, and we were unable to document the nature of the original problems. These two patients were excluded from our study, and we include 100 consecutive cases after phacoemulsification was performed in which we could determine the characteristics of the original retinal detachments. Surgery was performed on 98 of these eyes. Two eyes were considered inoperable because of massive periretinal membrane formation.

RESULTS

The 89 patients ranged from 20 to over 70 years of age (Table 1). Most patients were over age 60 years; 64 (72%) were male, and the right eye was involved in 59 (59%) of the cases. In 12 of the eyes we were able to document vitreous loss. A premature breakage of the posterior lens capsule occurred in at least eight. The posterior capsule was intact in only three of these eyes, and intraocular lenses were present in 19 eyes.

TABLE 1

AGES OF PATIENTS WITH RETINAL DETACHMENTS

Age (yrs)	Patients (%)
20–29	1
30–39	1
40–49	12
50–59	22
60–69	38
70+	26
Total	100

More than one half of the retinal detachments occurred within a year of phacoemulsification (Table 2); 33 of the detachments were total, and 12 were subclinical in that no visual field defect was present. The macula was involved in the detachment in two thirds of the cases. Significant preretinal membranes were noted preoperatively in 33 eyes; in eight, frank massive periretinal membrane formation was evident. In 59 of the eyes, multiple breaks were observed and in only four eyes were no breaks detected during surgery (Table 3). In 92 of the eyes vitreous traction was associated with at

TABLE 2

TIME OF RETINAL DETACHMENT AFTER PHACOEMULSIFICATION

Time	No. of Eyes
0–3 mos	31
3–6 mos	8
6–12 mos	21
1 yr +	32
Unknown	8
Total	100

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TABLE 3

CHARACTERISTICS OF RETINAL TEARS

Characteristic	No. of Eyes
Number	
Single	37
Multiple	59
None definite	4
Type	
At least one small tear with traction	60
At least one large horseshoe	32
Inferior dialysis 90 degrees or greater	4
No traction (small and round)	4
Location	
Quadrant	
Superior temporal	65
Inferior temporal	36
Superior nasal	35
Inferior nasal	13
Anteroposterior	
At least one at equator	45
More posterior	2

least one tear, and in only four eyes were all tears unassociated with vitreous traction. Most tears were small and located anteriorly. Large tears were observed in 36 eyes, and at least a single tear was evident at the equator or more posteriorly in 47 of the cases.

An analysis of these retinal breaks related to the time of the detachment revealed some interesting differences (Table 4). Detachments that occurred soon after surgery tended to be associated with larger tears, and with the exception of giant tears, these breaks were located more posteriorly than retinal tears that occurred more than one year after surgery. Single breaks were observed more commonly in detachments occurring soon after phacoemulsification than those that occurred more than one year after the procedure. Similarly, the percentage of eyes with tears in the superior nasal quadrant increased with an increase in the time segment between cataract surgery and retinal detachment.

TABLE 4

CHARACTERISTICS OF TEARS RELATED TO TIME OF DETACHMENT

Characteristic	Time of Detachment 0–6 mos	Time of Detachment 1 yr +
Number		
Multiple	54%	65%
Size		
Small only	45%	65%
Location		
Anterior only	47%*	58%
Superior nasal quadrant	22%	58%

*Includes four giant tears (11%).

Complications of phacoemulsification were related more closely to the detachments that occurred soon after cataract extraction than those that occurred later. The incidence of vitreous loss in those that occurred soon after cataract extraction was 21% (eight of 39 eyes), but no known vitreous loss occurred in the detachments that appeared more than one year after cataract surgery. Vitreous loss was associated with all four giant tears in this series. All giant tears occurred soon after phacoemulsification and were located inferiorly.

Reoperations were performed in four of the 75 eyes in which a 360-degree buckle was the initial procedure (Table 5). In three of these cases a vitrectomy combined with a second scleral buckling procedure was attempted, and a simple modification of the encircling element was used in the fourth. The overall success rate in this group of eyes was 93% (91 of 98 eyes). The success rates by procedure

TABLE 5

PRIMARY SURGICAL PROCEDURES FOR RETINAL DETACHMENT

Procedure	No. of Eyes
Transconjunctival cryothermy	12
Segmental buckle (90 degrees +)	8
360 degree buckle (exoplant)	75
Vitrectomy + buckle	3
Total	98

TABLE 6

REATTACHMENT RATES AFTER RETINAL DETACHMENT SURGERY*

Procedure	Percent
Transconjunctival cryothermy	100
Segmental buckle	100
360 Degree buckle	92
Vitrectomy + buckle	17
Overall success rate 93%	

*Ninety-eight primary and four secondary operations.

are listed in Table 6. All failures were caused by massive periretinal membrane formation. Preretinal membranes of significance were noted in all seven of these failures preoperatively, and in five of the seven failures frank massive periretinal membrane formation existed preoperatively. Postoperative visual acuities are listed in Table 7.

DISCUSSION

The characteristics of the retinal breaks and the configurations of retinal detachments after phacoemulsification are similar to those after intracapsular surgery. Such aphakic detachments are characteristically associated with a greater number of small, anterior, multiple breaks in the superior nasal quadrants than occur in phakic detachments.[2-4] Our data additionally suggest that detachments occurring soon after cataract surgery may be different than those that occur more than one year after cataract surgery. In eyes where detachment occurred soon after cataract surgery, tears tended to be single, larger, and more posteriorly located than those that occurred more than one year after phacoemulsification. The reasons for these apparent differences are not clear. Complications of cataract surgery may have played a significant role, because the percentage of eyes with known vitreous loss was considerably greater in the early detachments than those that occurred later. However, when eyes without a history of vitreous loss were considered separately, the number of small anterior breaks related to larger equatorial breaks, and the number of breaks in the superior nasal quadrant also increased with the time of detachment after phacoemulsification. Other possibilities for the apparent differences related to time include the presence of a pre-existing phakic retinal detachment and the existence of significant phakic vitreoretinal pathology that is aggravated by the sudden movement of the vitreous body forward. Tests for statistical significance were not performed in the evaluation of these data, and additional studies on more eyes will be required to prove that a difference between early and late aphakic detachments does exist.

Although the surgery for detachment after phacoemulsification is frequently more difficult because of problems in viewing the peripheral fundus,[1] the suc-

TABLE 7

VISUAL RESULTS AFTER SUCCESSFUL REATTACHMENT SURGERY

Before Surgery		After Surgery			
Visual acuity	No. of Eyes	6/7.5+ (20/25+)	6/9(20/30)–6/15(20/50)	6/18(20/60)–6/60(20/200)	6/120(20/400) and less
6/4.5(20/15)–6/7.5(20/25)	14	13	1		
6/9(20/30)–6/15(20/50)	13	4	8	1	
6/18(20/60)–6/60(20/200)	11	2	5	4	
6/120(20/400) and less	53	3	20	19	11

cess rates in this group of eyes compare favorably to previously published data on detachment surgery after intracapsular cataract extraction.[2,4,5]

SUMMARY

We reviewed the characteristics of 100 retinal detachments after phacoemulsification. The characteristics of retinal breaks and the configurations of the detachments were similar to cases of retinal detachments after intracapsular surgery. Evidence indicates that detachments occurring within six months of cataract surgery appeared to be statistically different from those occurring more than one year after phacoemulsification. Those detachments occurring within six months after cataract surgery tended to be more phakic in nature than those occurring relatively late. The surgical reattachment rates in these eyes compared favorably to previously published figures on the repair of retinal detachment after intracapsular surgery.

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CONGENITAL FIBROSARCOMA METASTATIC TO THE CHOROID

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Congential fibrosarcoma is a rare, rapidly growing soft tissue tumor with a tendency for local recurrence. There are few documented cases of congenital fibrosarcoma resulting in distant metastasis, and in none was the eye a site of metastatic involvement.

We report herein the clinical, histopathologic, and electron microscopic features of a congenital fibrosarcoma that metastasized to the eye two years after a primary tumor in the lower extremity. To our knowledge this is the first reported case of metastatic congenital fibrosarcoma to the eye.

CASE REPORTS

This boy was born spontaneously on Aug. 25, 1974, after normal full-term pregnancy and delivery. The APGAR Score was 9 at five minutes and the baby's weight was 2.230 kg. The entire lower left limb was grossly enlarged (Fig. 1). The child was brought to us on Aug. 27, 1977, and after examination and a clinical diagnosis of soft tissue sarcoma, a biopsy was performed. The histopathology was that of undifferentiated sarcoma. Chemotherapy begun Sept. 11, 1974, consisted of four doses of vincristine for five weeks, and a five-day course of cactinomycin (Actinomycin-D) and a cyclophosphamide. Within four weeks the white blood cell count dropped precipitously and he developed staphylococcal septicemia, for which he was treated with appropriate antibiotics. Chemotherapy failed to arrest further tumor growth, and a hindquarter amputation was done on Oct. 18, 1974. Investigation for evidence of metastasis including chest x-rays, skeletal survey, bone scan, total body gallium scan, and bone marrow biopsy were all negative. Indirect ophthalmoscopy performed at that time revealed a normal fundus. The patient had an uneventful recovery and was discharged on Nov. 18, 1974. He was followed up by the pediatric oncology service.

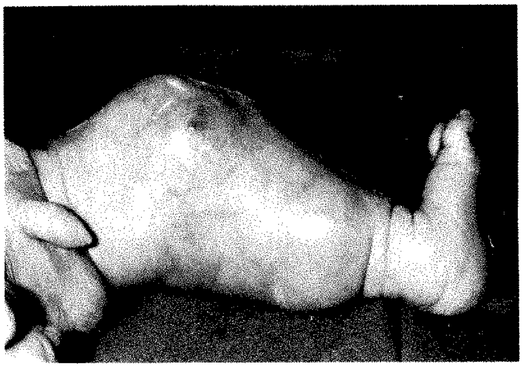

Fig. 1 (Rootman and associates). The left leg shortly after birth shows gross enlargement.

At 1 year of age the baby was readmitted for investigation of multiple dermal lesions that on biopsy proved to be benign angiomata. Chest x-rays and bone scan were normal.

In April 1977, the patient was seen elsewhere because of the recent onset of redness in the left eye. A shallow anterior chamber, sluggish pupil, and retinal detachment were found. The patient was referred to us for further examination and treatment. Bone marrow examination and bone, liver, and spleen scan were within normal limits. Examination under anesthesia carried out on April 20, 1977, revealed a shallow anterior chamber and engorged scleral vessels. The most striking finding was a solid mass involving the entire nasal half of the left eye with a shallow detachment of the overlying retina and an associated serous detachment of the temporal retina (Fig. 2). The lesion was completely transilluminable and ultrasound examination performed at that time identified a solid tumor with small cystic spaces that were estimated to be 1 to 2 mm apart. Fluorescein angiography was inconclusive. The clinical impression and the ultrasound diagnosis were believed to be consistent with hemangioma or metastatic fibrosarcoma.

On May 9, 1977, the child was operated on at which time a sclerostomy revealed a solid mass. An enucleation was performed. A nodular mass was noted surrounding the optic nerve and additional tissue was removed from the orbit.

Histopathologic examination confirmed tumor ex-

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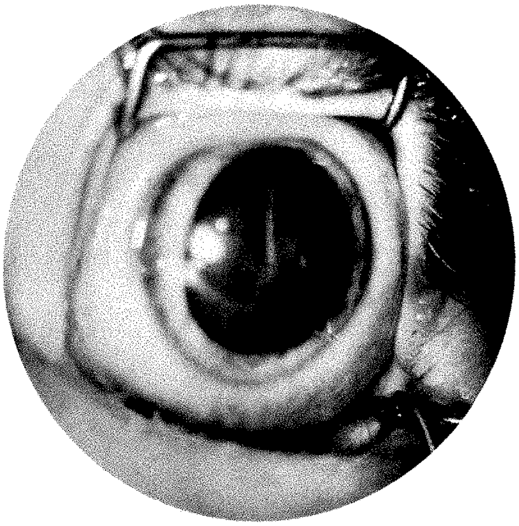

Fig. 2 (Rootman and associates). The left eye on April 20, 1977, shows a solid choroidal and overlying shallow retinal detachment.

tension beyond the globe, and computed tomographic scan suggested that there might be some minimal residual tumor in the inferior orbit. Because there was no evidence of systemic metastasis, an exenteration of the left orbit was performed on May 27, 1977. Pathologic examination showed no residual tumor in the orbit. The boy is alive and well 1½ years after exenteration.

RESULTS

The left leg was 19 cm in length, disarticulated at the hip, and deformed considerably by the large soft tissue mass. The tumor measured 10 × 7.5 × 5 cm in depth, was unencapsulated, displaced, and involved muscle and soft tissue. The tumor extended to but did not appear to infiltrate the skin, and joint involvement was not evident. Hemorrhagic and focal areas of cystic necrosis were present within the tumor and involvement of bone was noted.

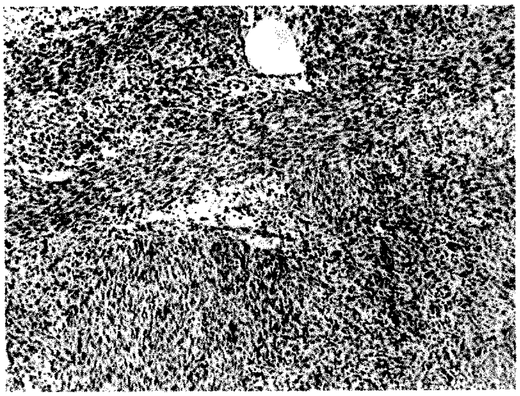

Fig. 3 (Rootman and associates). A compact area of tumor that has intertwining bundles of numerous small spindle cells (hematoxylin and eosin, ×97).

The tumor in the leg was highly cellular, moderately vascular, and had both a compact and loose fascicular architecture. In the dense zones were interwoven bundles of spindle cells (Fig. 3); this pattern persisted but was less obvious in the looser zones of the tumor. In some foci collagen formation was present as evidenced by fuchsinophilia on Masson trichrome staining. Here the tumor was less cellular and had the appearance of a fibromatosis. The cells were mostly fusiform or spindle-shaped with tapered amphophilic or lightly eosinophilic poorly outlined cytoplasm. Cross striations were absent on PTAH and Masson trichrome staining. Nuclei were elliptical or spindled and mitoses, some atypical, were evident (12 mitoses per ten high power fields). In several areas in the tumor the cells were more round and had plump, pale eosinophilic cytoplasm. These areas did not show cross striations. Reticulin stains showed a fine pericellular meshwork in the dense fascicular zones and a less well-formed pattern in the loose zones. The tumor invaded bone and skeletal muscle and approached closely the resection margin. Lymph nodes from this site were not involved by tumor.

On macroscopic examination the left eye measured 22 mm horizontally, 22 mm vertically, and 23 mm in diameter anteroposteriorly. The cornea was 11 mm horizontally and 10 mm vertically. The optic nerve measured 3 × 3 mm. There was a transillumination defect between 6 and 11 o'clock. The globe was sectioned horizontally and contained a large, easily transilluminated tumor mass that had a fish-flesh appearance and measured 19 mm in length, 12 mm in depth, and 17 mm in width. An epibulbar lesion was adjacent to the optic nerve posteriorly.

On microscopic examination the globe was characterized by the presence of a large tumor mass occupying the entire choroid from the optic nerve head to the iris root (Fig. 4). This tumor mass was made up of an admixture of loose zones as well as some areas of concentrated cellularity (Fig. 5). In the areas of the dense cellularity was an intertwining fascicular arrangement of the tumor cells where they appeared to be elongated with strap-like eosinophilic cytoplasm. Toward the center of this area, the cells were more undifferentiated with round nuclei and abundant eosinophilic cytoplasm that was PAS positive (Fig. 6). The cytoplasm of some cells appeared engorged with dense eosinophilic PAS-positive globular deposits. These deposits were also evident as light orange conglomerates on PTAH staining and occasionally fuchsinophilic on Masson trichrome staining. The loosely arranged areas contained cells that varied in nuclear size, shape, and staining properties, but on the whole had smaller nuclei. Special stains with Masson's trichrome and PTAH failed to reveal definitive cross striations. The stroma surrounding the cells was fuchsinophilic and showed collagen deposition.

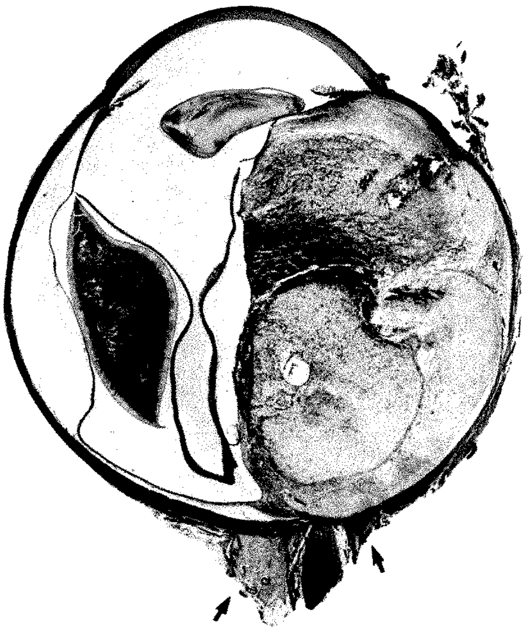

Fig. 4 (Rootman and associates). Gross specimen shows the tumor, the temporal serous detachment, and the orbital extension of the tumor around the optic nerve (arrows) (hematoxylin and eosin).

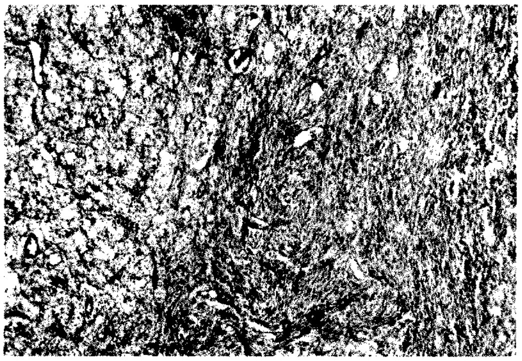

Fig. 5 (Rootman and associates). Microscopic section of the tumor. The tumor is composed of an admixture of loose zones as well as some areas of concentrated cellularity (hematoxylin and eosin, ×32).

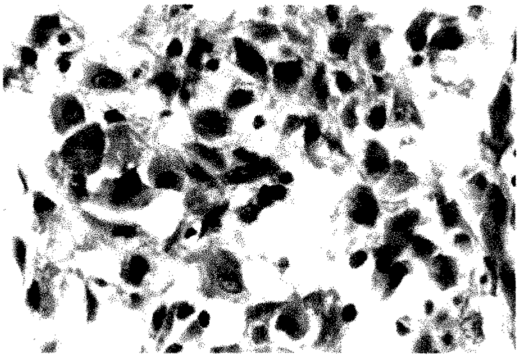

Fig. 6 (Rootman and associates). Central area of lesion. Note large, plump nuclei with abundant eosinophilic cytoplasm (hematoxylin and eosin, ×500).

Additionally, focal infiltrates of lymphocytes, plasma cells, and mast cells were evident. The ocular tumor was compared to the primary mass removed from the leg and was similar in character; however, the areas of plump cells with large amounts of eosinophilic cytoplasm were less evident in the primary specimen. The overlying pigment epithelium had undergone proliferation in focal areas and showed some drusen. There was degeneration of the outer layers of the overlying retina and the opposite side of the globe showed a serous retinal detachment. The tumor mass was noted to have extended via emissarial routes into the posterior episclera and the orbit surrounding the optic nerve (Fig. 4). The orbital tumor was cytologically identical. There was no evidence of invasion of the optic nerve.

The electron microscopic description was based on the appearance of the metastasis in the eye. The original biopsy specimen from the leg had an identical appearance. The tissue was fixed in formaldehyde and postfixed in 2.5% glutaraldehyde and prepared for electron microscopy. The tumor cells were spindle shaped and contained a moderate amount of cytoplasm, bound by a cell membrane, without basement membrane (Fig. 7). The nuclei were oval and had prominent nucleoli. Most cells were widely separated, leaving a large intercellular space. The cytoplasm was filled with extensive, rough endoplasmic reticulum, innumerable clusters of free ribosomes, and many mitochondria (Fig. 8, left). At the cell junctions, short, weakly marked desmosomes had formed with long gaps in between (Fig. 8, right). Fine fibrils branched off the surface of the extracellular deposits (Fig. 9, right) and appeared to give rise to bundles of fibers, one-fifth the diameter of collagen, with identical spacing but less well marked crossbanding (Fig. 9, left). The crossbands extended as fine wisps beyond the fibrils, and occasionally joined adjacent ones together. Collagen fibers were rare. The cytoplasm of the tumor cells was devoid of fibrils, and no Golgi apparatus was found. Lysosomal granules and pinocytotic vesicles were sparse.

DISCUSSION

Other investigators have described congenital fibrosarcomas,[1-3] and some of the more recent series[2,3] do not accept previously published cases as characteristic examples. Congenital fibrosarcoma most commonly affects the lower extremity, specifically the distal portion, whereas the adult form has a propensity for the thigh. There is a male prevalence of this rapidly growing locally aggressive tumor. The incidence of metastasis is low, varying from 0[2] to 8%[1,3] and has been noted in lymph nodes and lungs. The eye has never been involved as a metastatic locus.

Histologically, the tumors are composed of uniform spindle cells, similar to those of the adult type of fibrosarcoma, arranged in interweaving bundles, with the more differentiated lesion showing a characteristic herring bone pattern.

The differential diagnosis, histopathologically, should include aggressive fibromatosis, rhabdomyosarcoma, malignant schwannoma, angiosarcoma, and various other sarcomas. The distinction is not always easy to make. In a series of 31 cases,[1] only nine were initially reported as fibrosarcomas. Electron microscopy has indicated that the predominant cells are embryonic fibroblasts showing absence of basement membrane, collagen production, a highly developed Golgi apparatus, extensive rough endoplasmic reticulum, and poorly developed desmosomes between the tumor cells.[4]

The primary lesion in our case shows all the clinical features of a congenital fibrosarcoma. Histologically, it appears

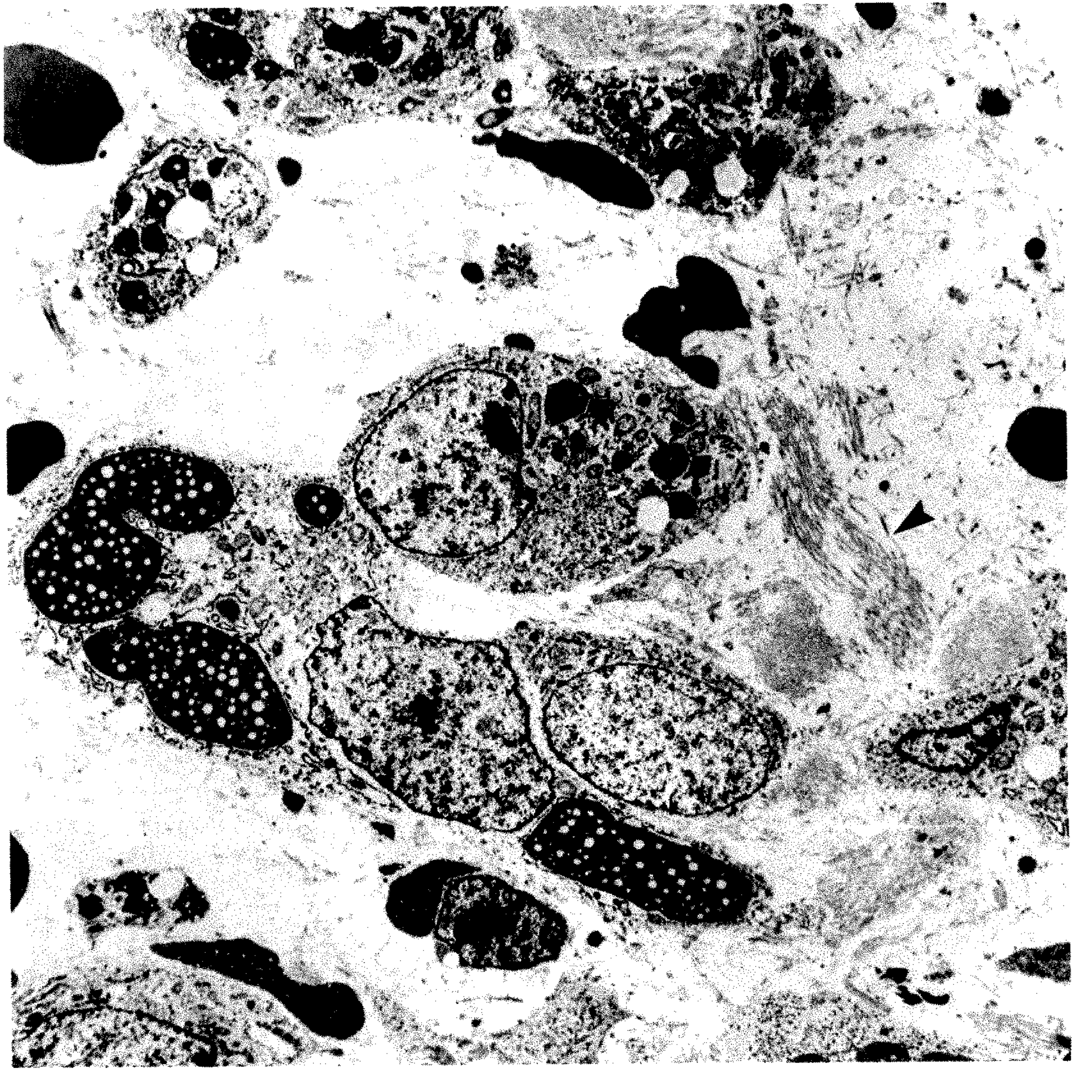

Fig. 7 (Rootman and associates). Low-power view of tumor. The cells are spindled and do not have a basement membrane. Intracisternal and extracellular electron-dense deposits and extracellular fibrils are present (arrow) (×3,000).

to be an exceptionally primitive and undifferentiated example of infantile fibrosarcoma. We believe the light microscopic and electron microscopic features indicate that the cell of orgin is a fibroblast.

The lack of basement membrane, presence of abundant rough endoplasmic reticulum, and poorly developed desmosomes indicate a fibroblastic origin.[5-12] We have not, however, noted a prominent Golgi apparatus. Additionally, a significant feature of our cells was the presence of pools of electron dense material endocisternally, within the cytoplasm, and extracellularly. This would appear to correlate with the light microscopic features of intracytoplasmic globular eosinophilic

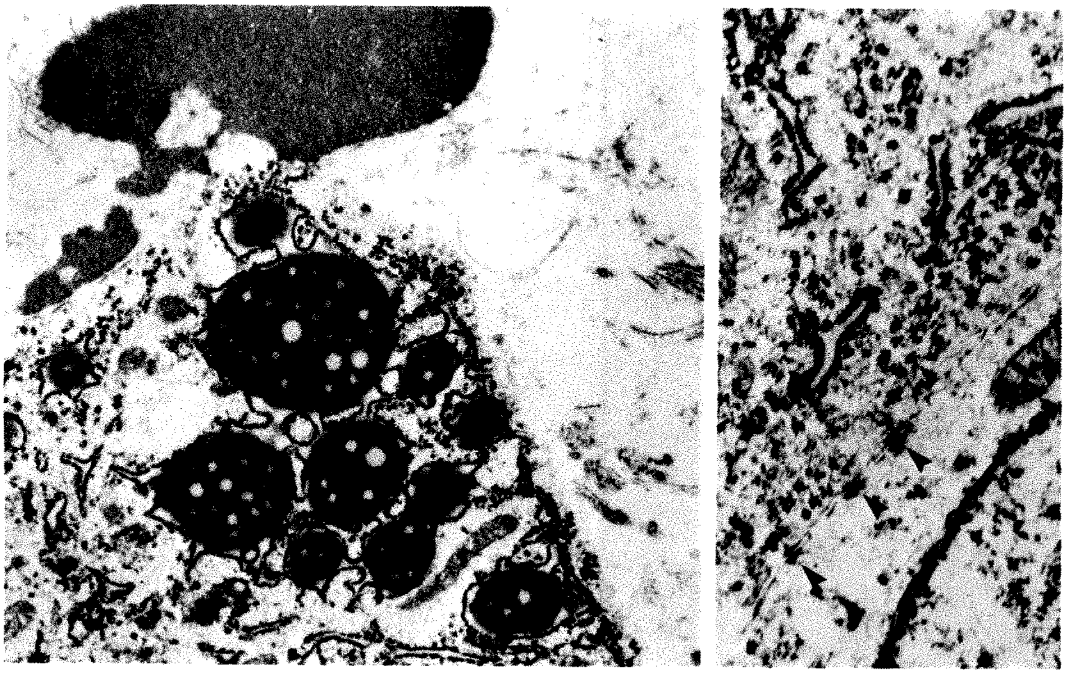

Fig. 8 (Rootman and associates). Left, Intracellular and extracellular dense deposits (×14,800). Right, Junctional complexes between two cells (arrows) (×31,000).

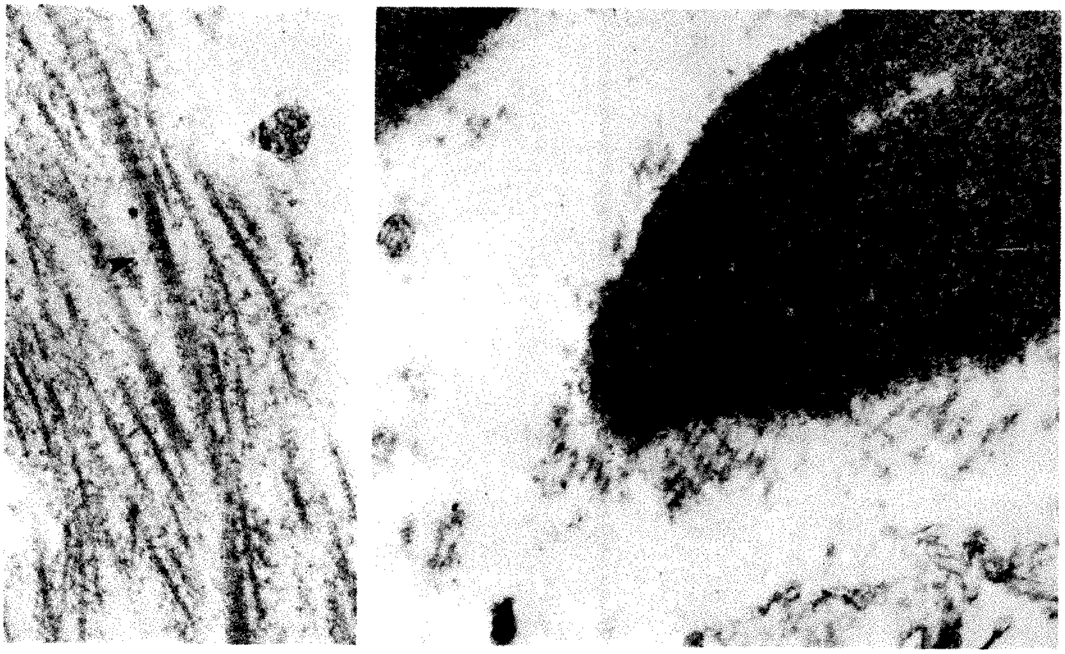

Fig. 9 (Rootman and associates). Left, Fibrils and one collagen fiber (arrow). Note cross striations in both (×53,800). Right, Fibrils streaming off dense deposits (×53,800).

PAS positive material. Occasionally we also noted lightly fuchsinophilic deposits intracellularly but more commonly fibrillar extracellular deposits were seen. We compared the cytology of these tumor cells with normal fibroblasts obtained from various patients by skin punch biopsy. Adult fibroblasts show an absence of basement membrane, dilated, rough endoplasmic reticulum and prominent Golgi apparatus, but also contain cytoplasmic fibrils and do not have endocisternal electron-dense material. The more immature fibroblasts from infants do have such material, though less marked than in our tumor. Jakobiec and Tannenbaum[5] described similar intracisternal material in their two orbital fibrosarcomas. Intracytoplasmic inclusions have been described in a number of the congenital fibromatoses both intracisternally and extracellularly.[13-15] Gonzales-Crussi[4] in an ultrastructural study of congenital fibrosarcoma also described an extracellular electron-dense material. The specific character and deposition of electron-dense material noted in our case appears to be unique in constellation, location, and amount. Additionally we were able to observe the origin of fibrillar material from the surface of these deposits, and the fibrils, with a thickness of one fifth of collagen fibers and weak crossbanding of similar periodicity, appeared to be a precursor of collagen. Thus, we believe the basic cell type is an immature fibroblast.

L. E. Zimmerman, M. D., and F. M. Enzinger, M. D., from the Armed Forces Institute of Pathology, Washington, D.C., reviewed our slides and agree with the diagnosis of poorly differentiated infantile fibrosarcoma. Dr. Raffaele Lattes, M. D., Columbia Presbyterian Hospital, New York, was not able to give a histogenetic classification and considered the ocular tumor to be an immature poorly differentiated metastasis from the lower limb primary.

SUMMARY

A 2½-year-old boy developed a choroidal metastasis from a congenital fibrosarcoma of the lower left limb that had been amputated shortly after birth. To our knowledge this is the first reported case of a congenital firbrosarcoma that metastasized to the choroid.

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INCIDENCE OF UVEAL MALIGNANT MELANOMA IN THE RESIDENT POPULATION OF ROCHESTER AND OLMSTED COUNTY, MINNESOTA

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Uveal malignant melanoma is the most common primary intraocular malignancy in the adult white population,[1,2] but is rare in blacks.[1,3,4] The general incidence of uveal malignant melanoma has been reported as high as 200 to 600 per million per year, [1,5-7] and as low as 4.9 per million per year [8] (Table 1). If the incidence of 200 to 600 per million per year were true, malignant melanomas of the uvea would be an overwhelming health problem. However, this high figure as cited by Yanoff and Fine[6] and others is the proportion of patients with uveal malignant melanoma in a large ophthalmology service and should not be interpreted as an incidence rate, which is the number of new cases per year in a specified resident population. The few published studies that have analyzed the occurrence of malignant melanoma in resident populations have reported incidence figures that are less than ten per million per year.

We report herein an investigation of the incidence of uveal malignant melanoma in the population of a well-defined community, Rochester and Olmsted County, Minnesota, for the 40 years from 1935 through 1974 and covers 2,700,000 person-years of experience (person years is the average population of the community [persons] multiplied by number of years of observation).

TABLE 1

PUBLISHED INCIDENCE OF UVEAL MALIGNANT MELANOMA IN THE WHITE POPULATION

Source	Incidence/million
1927 Davenport[7]	200-600
1959 Dorn and Cutler[9]	5.2
1963 Jensen[10] (Denmark)	7.5
1973 Ganley and Comstock[11]	6.6
1975 Cutler and Young[12]	5.6
1977 Shammas and Watzke[8]	4.9

MATERIAL AND METHODS

Records on uveal malignant melanoma, including melanomas of the iris, ciliary body, and choroid, of residents of Rochester and Olmsted County were used. For major illnesses, including malignancies, most of the medical care of Olmsted County and the Rochester population was provided by the Mayo Clinic. However, diagnoses from other health care institutions in and around Olmsted County, as well as all death certificates, are regularly entered into a central registry, which is maintained at the Mayo Clinic.

Each year, about half of the local population is seen at the Mayo Clinic, its affiliated hospitals, or through home visits, for which diagnoses are recorded. We reviewed all cases in which the diagnosis of uveal malignant melanoma was made within the 40-year period from 1935 through 1974. Additionally, the University Hospital and the Veterans Administration Hospital in Minneapolis were contacted so that any Rochester-Olmsted County resident with malignant melanoma seeking care at that institution could be identified. Further information was obtained from autopsy records. In recent decades, autopsy has been performed on

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about 70% of all local patients who have died, whether at home or in one of the hospitals, and these results have been incorporated into the central registry. However, the autopsy incidence of malignant uveal melanoma is not entirely accurate because ophthalmic pathologic study is not done in all cases.

Cases were retained only if the patient had been a resident of the city or county at the time of initial diagnosis and if the diagnosis of malignant uveal melanoma was confirmed through re-examination of the tissue by one of us (R.J.C.). If residence in Rochester or Olmsted County had been established to facilitate treatment of an illness, the case was excluded. For completeness, we also reviewed our files for patients whom we presently are following up who have the clinical diagnosis of presumed uveal malignant melanoma. All patients who had malignant melanoma of the posterior choroid in the study period had enucleation.

RESULTS

From Jan. 1, 1935 to Dec. 31, 1974, uveal malignant melanoma was diagnosed in 15 patients (five males, ten females) involving the residents of the city of Rochester and Olmsted County. The average annual rate per million was five for males, nine for females, and seven for both sexes combined. The age at diagnosis ranged from 17 to 72 years, with a mean of 54.5 years and a median of 61 years (Table 2). The rate per million for both sexes combined was three for those less than age 50 years and 21 for those age 50 years or older.

Histopathologically, the choroid was the predominant site of origin in 11 cases, four of mixed type, six of spindle B, and one of spindle A; the iris was the site in one case (spindle A type); the iris and ciliary body were the sites in one case (spindle A type); and the choroid and ciliary body were the sites in two cases, both of mixed cell type (Table 3).

TABLE 2

AGE DISTRIBUTION OF PATIENTS WITH UVEAL MALIGNANT MELANOMA*

Age (yrs)	No. of Cases	Ages (yrs)
0-9	0	—
10-19	1	17
20-29	1	25
30-39	1	32
40-49	2	41, 41
50-59	1	51
60-69	4	61, 61, 64, 67
70-79	5	70, 71, 71, 72, 73
Total	15	

*Median age, 61 years.

DISCUSSION

According to Dorn and Cutler,[9] the annual incidence of ocular malignant melanoma in the United States in 1947 was 5.2 per million per year. In an updated study by Cutler and Young,[12] the 1970 rate was calculated to be 5.6 per million per year. In 1963, Jensen[10] reported the incidence of uveal malignant melanoma to be about 7.5 per million per year for the period 1943 through 1952.

Shammas and Watzke[8] reported that the annual incidence of uveal malignant melanoma was 4.9 per million per year; the figure being based on 41 cases reported in the state of Iowa from 1969 to 1971. From six cases of choroidal and ciliary body melanomas that were noted in Washington County, Maryland, from 1956 through 1965, Ganley and Comstock[11] estimated an annual incidence rate of 6.6 cases per million per year.

In our study, the overall incidence in the resident population of Rochester and Olmsted County was calculated as seven per million per year. The incidence in the population age 50 years or older in our study is 21 per million per year. Other

TABLE 3

HISTOPATHOLOGIC FEATURES OF UVEAL MALIGNANT MELANOMA

		Location			
Cell Type	No. of Cases	Iris	Choroid	Iris and Ciliary Body	Choroid and Ciliary Body
Spindle A	3	1	1	1	0
Spindle B	6	0	6	0	0
Mixed	6	0	4	0	2

investigators have reported higher incidence rates with increasing age. In the population age 30 years or older, Ganley and Comstock[11] reported an incidence rate of 13 per million per year. In Jensen's[10] study from Denmark, the incidence rates of choroidal and ciliary body melanomas were highest for persons between the ages of 60 and 80 years. In Mork's[13] study from Norway, the highest incidence rates were in the age group from 70 through 79 years.

SUMMARY

Uveal malignant melanomas were diagnosed in 15 patients in the resident population of Rochester and Olmsted County, Minnesota, from 1935 through 1974. There were five males and ten females. In this well-defined community, the annual incidence of uveal malignant melanoma for the 40-year period was calculated to be seven per million per year. The incidence in the resident population under age 50 years was three per million, whereas for those age 50 years or older, it increased to 21 per million per year.

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OCULAR MANIFESTATIONS OF THE 1976 RUBELLA EPIDEMIC IN JAPAN

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An extensive epidemic of rubella occurred throughout Japan from 1975 to 1977. Six patients from ages 7 to 26 years with acquired rubella and epithelial keratitis characteristic of the disease were seen by us in 1976.

Reports concerning acquired rubella keratitis are rare; one by Zade[1] and another by Smolin.[2] Smolin reported a case of rubella with bilateral epithelial corneal erosions localized mainly in the central area of the cornea.

We describe herein ocular symptoms of patients with rubella in this epidemic, particularly the characteristic clinical symptoms of keratitis among the young.

SUBJECTS AND METHODS

Seventy-nine patients with acquired rubella were examined by us in 1976 and 1977. In 1976, there were 76 cases from April 3 to June 25 and in 1977 only three cases from March 30 to June 15, 1977. All were outpatient cases, clinically diagnosed as acquired rubella. There were 38 males and 41 females, ages 1 to 29 years (Table 1).

TABLE 1

AGE DISTRIBUTION OF CASES OF RUBELLA

Age (yrs)	No. of Cases
1-5	17
6-10	35
11-15	17
16-20	1
21-25	6
26-29	3

Ocular examinations consisted of measurements of visual acuity where possible, inspection of the anterior segments of the eye, slit-lamp examination, and ophthalmoscopic examination. Facial rash and swelling of preauricular lymph nodes were also studied.

The initial ocular examinations were done on the day when the rash appeared or the next day (Table 2). Cases of rubella with keratitis were examined daily or every other day until the keratitis disappeared. Follow-up examinations of the cases without keratitis were done for ocular hyperemia, irritation, and photophobia. Of five patients without keratitis, two developed epithelial keratitis.

TABLE 2

INTERVAL BETWEEN ONSET OF SKIN RASH AND INITIAL OCULAR EXAMINATION

Interval (days)	No. of Cases
0	23
1	30
2	12
3	3
4-7	8
≥8	3

CASE REPORT

A 14-year-old girl was seen initially on April 3, 1976, complaining of photophobia and irritation of both eyes of one day's duration. One week before the onset of ocular symptoms, she developed fever, enlarged posterior cervical lymph nodes, and rash on the face, trunk, and limbs that had been diagnosed as rubella. Her younger sister also suffered from rubella on the same day.

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On examination her visual acuity was 6/6 (20/20) in both eyes. There was mild, bilateral hyperemia of the bulbar and palpebral conjunctiva. About 100 fine white punctate opacities were disseminated mainly in the central part of the cornea (Figure). Punctate opacities, limited to the epithelial layer, stained with fluorescein. No anterior chamber reaction was found. Neither swelling of the preauricular lymph nodes nor rash on the face was present. She was given topically applied 1% chloromycetin and 0.1% dexamethasone three times a day and an intramuscular injection of gamma globulin (150 mg/ml; 450 mg). By April 5, ocular irritation and photophobia had improved. Punctate corneal opacities not stainable with fluorescein were still present. Gamma globulin eye drops (30 mg/ml in physiological saline) were administrated to the right eye. All other therapy was discontinued. By April 7, the cornea of the right eye had become clear, but punctate corneal opacities were still present in the left eye. By April 10, keratitis of the left eye had disappeared.

Laboratory test results included: 518 × 10^4/mm^3 red blood cells; 25.9 × 10^4/mm^3 platelets; and white blood cells, 4,800/mm^3, with 57% polymorphonuclear leukocytes, 27% lymphocytes, 9% eosinophils, 1% basophils, and 6% monocytes. Erythrocyte sedimentation rate was 4 mm/hr. Serum immunoglobulins were: IgG, 1,470 mg/dl; IgA, 385 mg/dl; IgM, 225 mg/dl; and IgD, 13 mg/dl. Viral isolation from the conjunctival sac was negative. Antibody titer to rubella hemagglutination inhibition was 1:128 on April 5 and 1:256 on April 17, 1976.

RESULTS

Among 79 patients, 56 (70.9%) had mild catarrhal or follicular conjunctivitis. Palpebral hyperemia was more marked than bulbarhyperemia. Six patients (7.6%) had epithelial keratitis; fine punctate epithelial opacities were disseminated mainly in the central area of the cornea. No disease was evident in the anterior chamber or the fundus (Table 3).

Six cases with rubella keratitis were diagnosed in 1976. Four patients had epithelial keratitis at their first visit, and two patients had epithelial keratitis the first and fifth days after their first visit. Almost all these patients came on the day when their subjective ocular discomforts first appeared. Bilateral ocular pain, irritation, and photophobia were common complaints. The interval between the onset of rash and the appearance of keratitis was two to 13 days (Table 4). In many cases, keratitis developed about one week after the skin rash, and disappeared with-

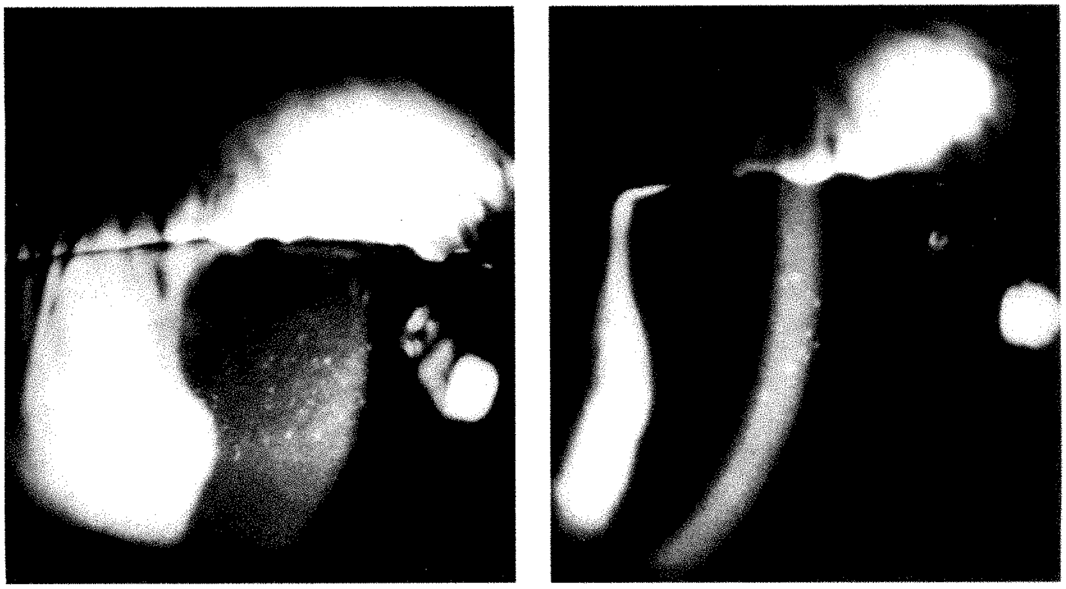

Figure (Hara and associates). Slit-lamp photographs showing rubella keratitis. Left, Fine punctate epithelial opacities centered on the middle part of the cornea. Right, White discrete opacities located in the epithelial layer of the cornea.

TABLE 3

INCIDENCE OF OCULAR SIGNS IN RUBELLA

Ocular Signs	No. of Cases	Percent
Conjunctivitis	56	70.9
Keratitis	6	7.6
Iridocyclitis	0	
Rash on the face	18	22.8
Swelling of preauricular lymph nodes	17	21.5

in one week with no sequelae. Keratitis was treated with gamma globulin eye drops three times a day except in the left eyes of two patients (Table 4, Cases 1 and 5). Punctate epithelial opacities, characteristic of keratitis, were disseminated in the central part of the cornea. These pinpoint-sized punctate lesions were about 0.2 to 0.05 mm in diameter, averaging about 0.1 mm, but varied in number per cornea from a few to about 100. These opacities were limited to the epithelial layer except in one case. Some stained faintly with fluorescein. Keratitis developed bilaterally with slight conjunctivitis, but without anterior chamber reaction.

DISCUSSION

From 1975 to 1977, an extensive epidemic of more than 2 million cases of rubella occurred throughout Japan after about a ten-year interval. There was also a rubella epidemic in Itami City, with a population of 140,000, of which 7,000 were afflicted with rubella from January to June 1976. This study was based on examinations of rubella in 79 cases between April 1976 and June 1977.

The prominent ocular symptom of this rubella epidemic was rubella keratitis. Of 79 cases, six had rubella keratitis. An analysis of the data revealed that the epidemic incidence of rubella keratitis was about 2.5% (two cases with rubella keratitis per 75 cases of rubella), because four of the patients had come directly to us with rubella keratitis.

Cases of rubella with keratitis commonly showed fine punctate opacities in the central area of the cornea. Smolin[2] reported a similar case of transient rubella keratitis in a 16-year-old boy, in which approximately 15 punctate lesions were visible on the cornea in the central area of the palpebral fissure. Punctate epithelial opacities disseminated mainly in the central area of both eyes may indicate rubella keratitis. The disease had a good prognosis because epithelial keratitis disappeared within one week without any sequelae.

The presence of similar epithelial keratitis has been reported during the course of rubeola and mumps.[3,4] In our cases, however, bilateral epithelial corneal lesions might be rubella keratitis. Sugges-

TABLE 4

RUBELLA WITH KERATITIS

Case No.	Initial Visit (1976)	Age (yrs)	Sex	Interval between Onset of Rash and Appearance of Keratitis (days)	Duration of Keratitis (days)*	
					R.E.	L.E.
1	4/3	14	F	8	4	7
2	4/14	14	F	11	5	3
3	4/19	7	F	7	5	2
4	4/27	9	F	2	2	2
5	5/11	13	F	7	3	3
6	5/11	26	F	13		

*The duration from its first documentation to its documented disappearance.

tive of the diagnosis of rubella were: (1) the recent exposure to rubella epidemic; (2) the absence of Koplik spots; (3) the typical appearance of rash; (4) the typical course of the disease (the minimal febrile reaction, the lack of malaise, and early appearance of enlargement of the posterior cervical lymph node); and (5) establishment of serologic proof in some cases.

Topical administration of gamma globulin[5] was used to treat acquired rubella keratitis. Further study seems necessary in evaluating the efficacy of gamma globulin for the treatment of rubella keratitis.

SUMMARY

Of 79 cases of rubella during the 1976-1977 rubella epidemic in Japan, 56 (70.9%) had mild conjunctivitis. Six (7.6%) showed epithelial keratitis, but the epidemically estimated incidence of keratitis was about 2% (two cases with rubella keratitis per 75 cases of rubella). No evidence of disease of the anterior chamber or the fundus was observed. Characteristic features of keratitis in juvenile rubella were fine punctate epithelial opacities mainly disseminated in the central area of the cornea. The punctate lesions were about 0.1 mm in diameter on an average, but varied in number from a few to about 100. In many cases, keratitis developed about one week after skin rash and disappeared within one week with no sequelae.

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PRERETINAL ARTERIAL LOOPS AND RETINAL ARTERIAL OCCLUSION

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The preretinal arterial loop, first described by Liebreich in 1871,[1] is a congenital vascular anomaly that originates from a main branch of the central retinal artery on the optic disk.[2] These arterial loops, which are usually unilateral, are more common than preretinal venous loops.[3] Although they are usually asymptomatic, three cases of preretinal arterial loops associated with branch retinal arterial occlusion have previously been described.[4-6] We report herein a case of unilateral branch retinal artery occlusion in a patient with bilateral preretinal arterial loops.

Case report

A 19-year-old man came to the emergency room after noting loss of vision in the upper visual field of the right eye upon awakening that morning. There was no history of trauma, earlier visual dysfunction, headache, or any systemic disease.

Ocular examination revealed a visual acuity of 6/6 (20/20) in both eyes. The pupils were equal, round, and reactive to light, but there was an afferent pupillary defect in the right eye. Slit-lamp examination was normal in each eye and applanation pressure was 16 mm Hg in both eyes.

Ophthalmoscopy of the right eye revealed an arterial trunk which passed from the optic disk, extended for 1 to 2 mm into the vitreous cavity and abruptly returned to the optic disk to form the inferior papillary artery. There was nerve fiber layer edema in the distribution of the inferior papillary artery (Fig. 1), consistent with a branch retinal artery occlusion. There were no emboli or plaques within the vessel. Intravenous fluorescein angiography showed no filling of the preretinal loop throughout the study and segmentation of the blood column within the inferior papillary artery (Fig. 2).

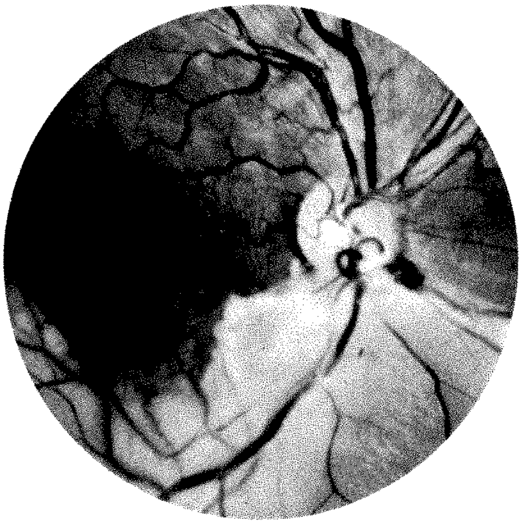

Fig. 1 (Brown and associates). Preretinal arterial loop with associated nerve fiber layer edema in the distribution of the inferior papillary artery in the right eye. The small dark object adjacent to the inferonasal disk is retinal hemorrhage.

Examination of the left eye revealed a pulsating preretinal vascular loop over the disk, extending 3 mm into the vitreous cavity (Fig. 3). Fluorescein angiography of this eye disclosed filling of the superior papillary artery before the appearance of background choroidal fluorescence (Fig. 4, top left). As the angiogram progressed we could see that the superior fundus and the macula were supplied by this large superior cilioretinal artery and a smaller inferotemporal cilioretinal artery (Fig. 4, top right). Fluorescein dye appeared in the loop shortly after filling of the cilioretinal arteries, but approximately 3.5 seconds elapsed before the loop demonstrated fluorescence as intense as within the cilioretinal arteries (Fig. 4, top right, bottom left and right), indicating a relatively slow flow through this arterial system. Inferonasal choroidal background fluorescence and inferior papillary retinal arterial filling were absent until the arterial loop was filled with

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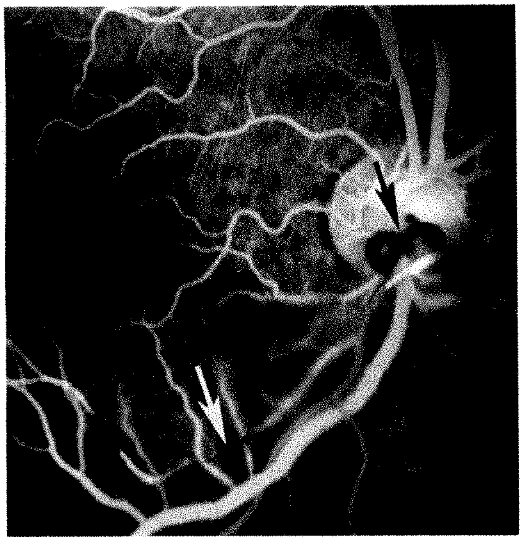

Fig. 2 (Brown and associates). Intravenous fluorescein angiography of Figure 1. No filling is seen within the arterial loop (black arrow) and segmentation of the arterial blood column is present within one of the branches of the inferior papillary system (white arrow).

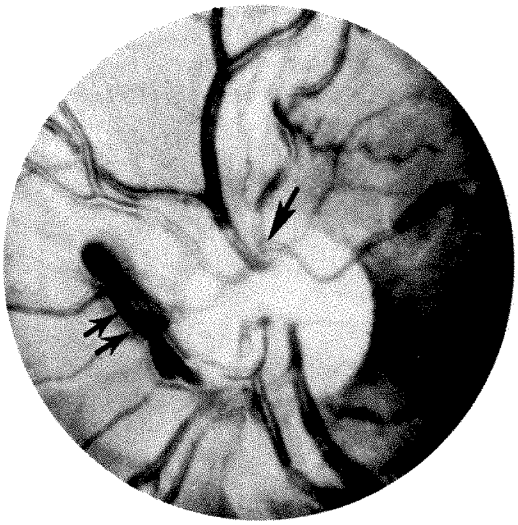

Fig. 3 (Brown and associates). Spiral preretinal arterial loop extending into the vitreous and then returning to the optic disk in the left eye (double arrows). The superior retina is supplied by a large branching cilioretinal artery with its origin (single arrow) at the superior disk margin.

fluorescein dye (Fig. 4, top right, bottom left and right), suggesting that blood supply to both of these areas was derived from the preretinal arterial loop.

Results of complete physical examination were normal, as were results of laboratory tests for cardiovascular and collagen disease. An extensive hematologic examination, including hemoglobin electrophoresis, disclosed a nonspecific mild rise in factor VIII procoagulant activity.

No therapy was undertaken and the patient was next seen months later. Visual acuity in the right eye was 6/5-2 (20/16)-2 at this time. The nerve fiber layer edema had subsided and the preretinal arterial loop appeared as a subtle white ghost vessel, devoid of blood. Fluorescein angiography (Fig. 5) showed antegrade filling of the inferior papillary artery from a newly demonstrable cilioretinal artery originating from the inferonasal aspect of the optic disk. No fluorescein was seen within the former arterial loop throughout the study. Visual field examination disclosed an absolute superior defect (Fig. 6).

On the most recent examination, eight months after the occlusion in the right eye, the cilioretinal artery supplying the inferior retina was more prominent and the blood column had increased in size (Fig. 7). Fluorescein angiography showed filling of the superior and inferior cilioretinal system before the filling of the small central retinal arterial branch supplying the fovea (Fig. 7, center and bottom). Despite the early filling of the inferior arterial supply to the retina, the arteriovenous transit time was delayed in comparison to the superior retina. Visual acuity was 6/5-2 (20/16)-2 in this eye and the absolute superior visual field defect remained. The left eye was unchanged from the previous visit.

DISCUSSION

In the one case of a preretinal arterial loop described histologically, the vessel originated from a main branch of the central retinal artery and continued as a loop within the course of that branch.[2] Embryologically, these loops may be derived at about the 100 mm stage from within the area of Bergmeister's papilla and have no connection with the hyaloid artery.[7]

The loops may be single or take one or more spiral turns, as shown in our patient. They may enter and exit at the disk or enter by the disk and continue into the retina as a branch vessel.[8]

Preretinal vascular loops had been reported in only 63 eyes by 1953.[3] Of these, 52 (83%) eyes contained arterial loops and 11 (17%) contained venous loops. Bilater-

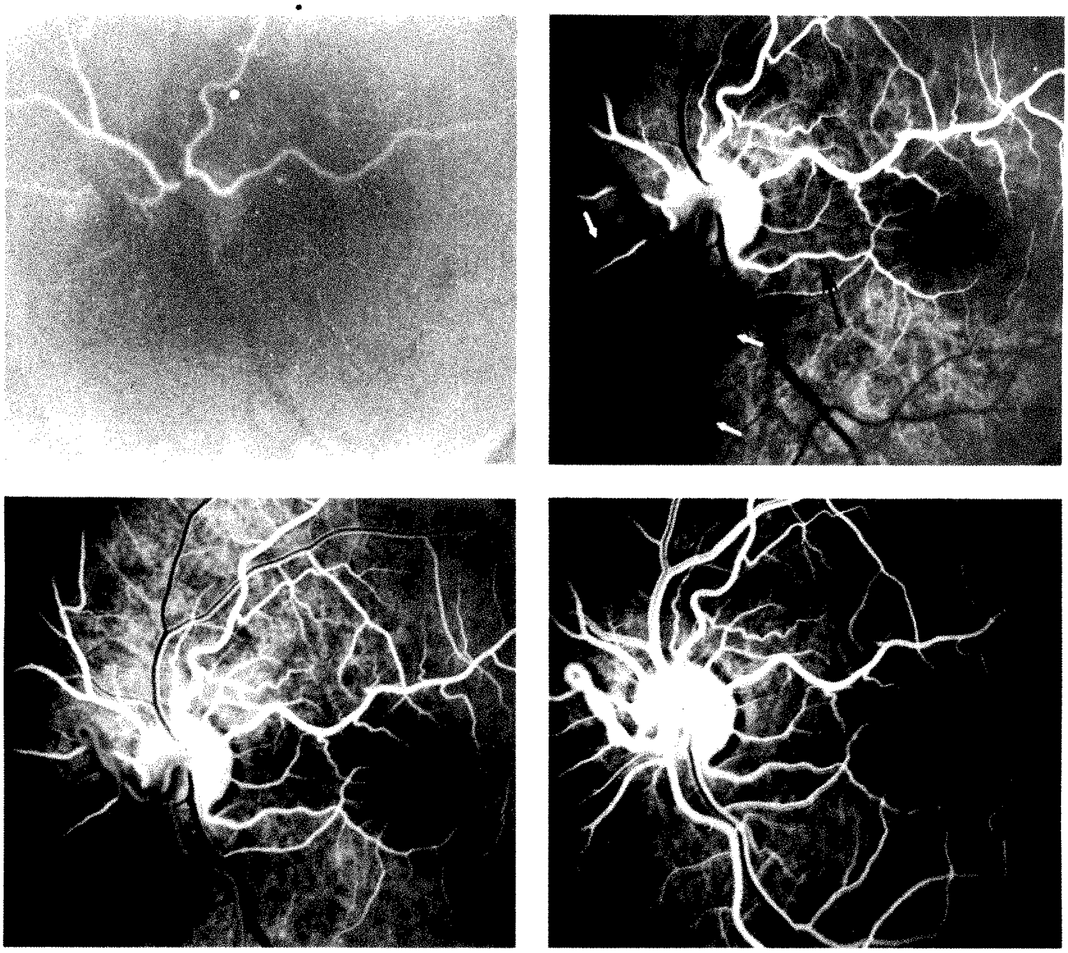

Fig. 4 (Brown and associates). Fluorescein angiogram of the left eye. Top left, Filling of the superior papillary cilioretinal artery before the appearance of choroidal fluorescence. Top right, The superior papillary and inferotemporal (black arrow) cilioretinal arteries are well filled. Dye is faintly appearing within the loop, but the inferior papillary artery and the inferonasal choroid (outlined by white arrows) are relatively hypofluorescent. Bottom left, Hyperfluorescence is more prominent within the prearterial loop and inferotemporal retinal artery. The inferonasal choroid is still relatively hypofluorescent. Bottom right, the preretinal arterial loop is completely filled with fluorescein dye, as now are the inferior retinal arteries and the inferonasal choroid. The progression of blood through the loop and then the inferior retinal arteries and the inferonasal choroid suggests that the loop may supply blood to both the retina and the choroid.

al arterial loops were present in about 8% (4 of 48) of patients with preretinal arterial loops. Arterial occlusion was associated in 4% (2 of 52) of eyes with arterial loops.

Oxilia[9] estimated that 70% of preretinal arterial loops involved the inferior retinal vessels, and 30% involved the superior retinal vessels. In this case, as well as in the three previously reported cases of arterial occlusion associated with loops,[4-6] only the inferior retinal vessels were involved. One of the four patients had visual acuity of counting fingers, although the others maintained visual acuity of 6/6 (20/20) or better in the involved eye. If the involved vessel supplies a significant por-

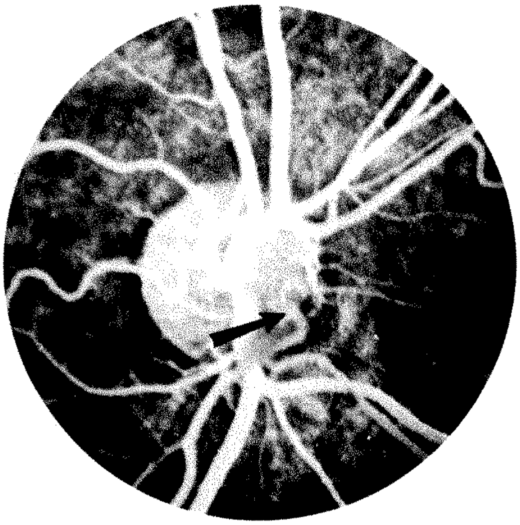

Fig. 5 (Brown and associates). Right eye three months after inferior papillary arterial occlusion. Fluorescein angiography discloses a small cilioretinal vessel (arrow) now supplying the inferior retina. This vessel could not be visualized at the initial examination (Figs. 1 and 2).

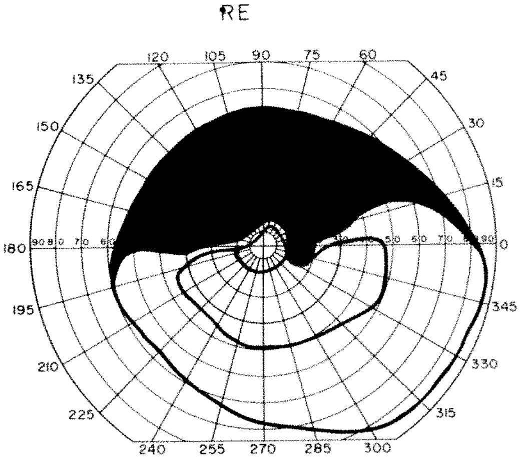

Fig. 6 (Brown and Associates). Visual field of the right eye, showing an absolute superior altitudinal field defect.

tion of the fovea, the prognosis for good visual acuity diminishes.

The cause of occlusion is thought to be kinking or twisting of the loops, with subsequent obstruction.[6] Although this may have been the situation in our case, the precise mechanism is not clear. Flow through the loop may also be somewhat slow, as evidenced by delayed filling of the inferior papillary artery on fluorescein angiography of the left eye in our patient. Presently detectable systemic diseases associated with retinal arterial occlusion appear not to have a significant role. Atherosclerotic changes are doubtful, as three of the four patients with occlusions have ranged in age between 19 to 22 years. Emboli have not been observed in any of these cases.

This case shows that a major portion of the retinal arterial blood supply in each eye is cilioretinal in origin. Although it is impossible to know whether the occluded arterial loop in the right eye was cilioretinal, the probable collateral vessel supplying the inferior retina months after the occlusion is characteristically cilioretinal. Most of the retina in each eye is rarely supplied by cilioretinal arteries; Collier[10] found only one such case in 250 eyes with cilioretinal arteries. In the right eye of our patient the foveal vision was spared by a small branch of the central retinal artery, whereas much of the rest of the fundus was supplied by a cilioretinal system. Central retinal or branch retinal artery occlusion with cilioretinal foveal sparing[11] is more common.

The presence of an artery that supplies both the retina and the choroid, as may be the case in the left eye of our patient, is rare except for retinochoroidal anastomoses with disciform lesions of the macula and after focal argon laser photocoagulation. Anastomoses between retinal and ciliary circulations[12,13] and between the cilioretinal and choroidal systems[10] have been described. We are currently attempting to determine whether preretinal arterial loops are, other than by chance, associ-

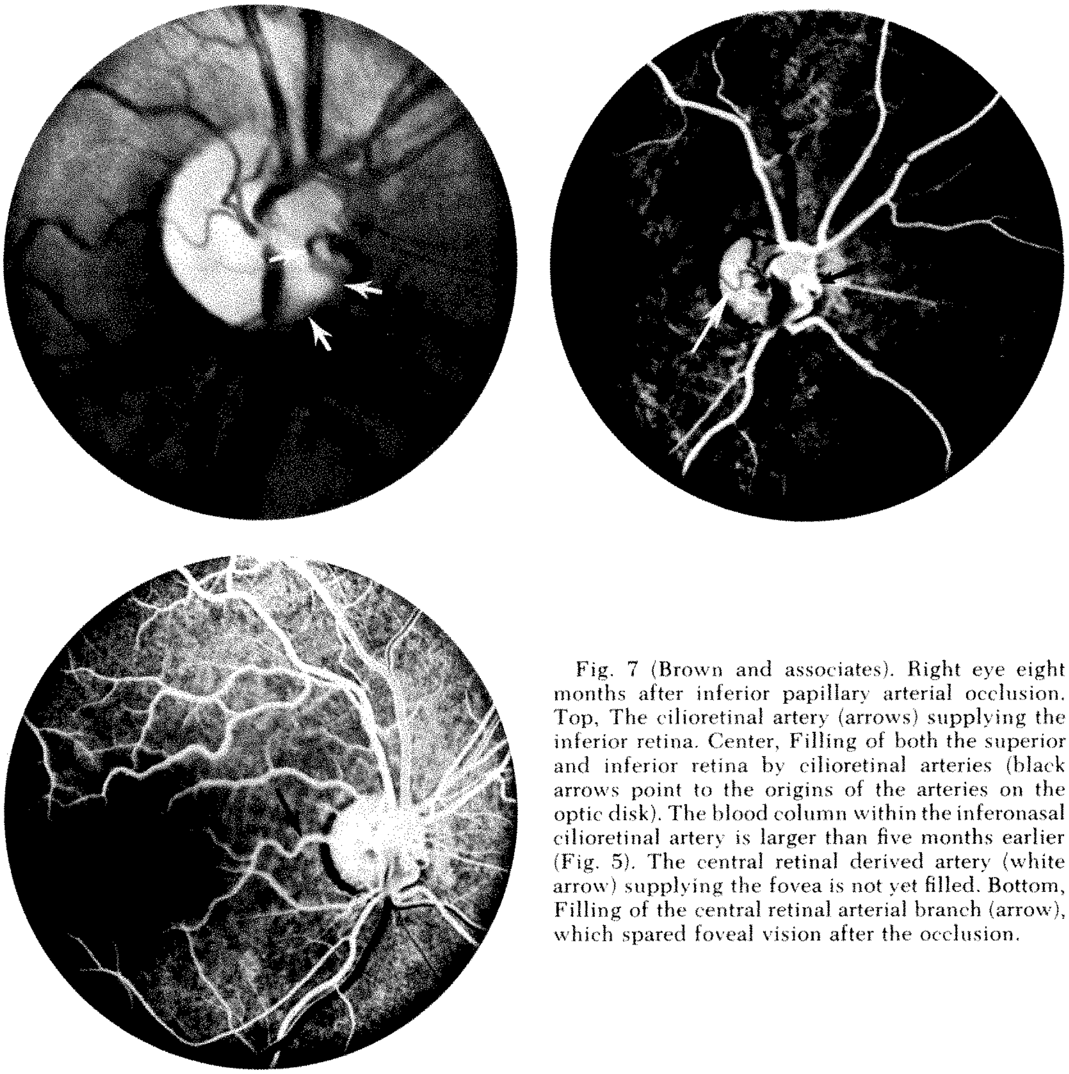

Fig. 7 (Brown and associates). Right eye eight months after inferior papillary arterial occlusion. Top, The cilioretinal artery (arrows) supplying the inferior retina. Center, Filling of both the superior and inferior retina by cilioretinal arteries (black arrows point to the origins of the arteries on the optic disk). The blood column within the inferonasal cilioretinal artery is larger than five months earlier (Fig. 5). The central retinal derived artery (white arrow) supplying the fovea is not yet filled. Bottom, Filling of the central retinal arterial branch (arrow), which spared foveal vision after the occlusion.

ated with vessels supplying both retina and choroid.

SUMMARY

A 19-year-old man had bilateral congenital preretinal arterial loops emerging from the optic disk into the vitreous cavity. The loop in the right eye was associated with an inferior papillary arterial occlusion. Extensive examination to determine a systemic cause of the occlusion was negative; twisting or kinking of the loop may have had a role. The major arterial blood supply in each eye was cilioretinal in origin and in the left eye, the inferior preretinal arterial loop supplied blood to both the retina and the choroid.

In three previous cases and our case of preretinal arterial loops associated with branch retinal arterial occlusion, none had emboli or associated systemic factors.

All involved solely arterial supply to the inferior retina. The ages of three of the four patients ranged from 19 to 22 years.

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THE EFFECT OF PROLONGED INDIRECT OPHTHALMOSCOPY ON THE HUMAN EYE

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In recent years, several investigators have shown in experiments with laboratory animals that retinal photoreceptors and pigment epithelial cells may be permanently damaged by prolonged exposure to light.[1-11] Under strictly controlled laboratory conditions, light from an indirect ophthalmoscope focused for intervals of 15 to 60 minutes by a 20-diopter condensing lens onto the macular retina of rhesus monkeys has produced photic retinopathy. Such experiments have caused uncertainty regarding the tolerance of the human retina to prolonged light exposure, such as that of the newborn when exposed to phototherapy used in the management of hyperbilirubinemia.[12] Uncertainties also exist regarding the tolerance of the adult retina to high-intensity light from instrumentation used in clinical ophthalmology.[13] Because an integral part of any thorough clinical evaluation of the human eye involves examination with instruments equipped with bright-light sources, the question of light damage to the human retina is one of great practical concern, especially when prolonged repeated examinations with indirect ophthalmoscopy are done.

We report herein a study on the effects of high-intensity light on the human eye by using the standard light and power source of an indirect ophthalmoscope in an experiment designed to simulate an excessively prolonged clinical examination of the adult retina.

SUBJECTS AND METHODS

Patients included in the study had either a functioning eye that contained a tumor requiring enucleation (Table 1) or a totally blind eye with clear media (Table 2). The motivation and attitude of the patients were carefully appraised by the investigator. Seven patients with a positive attitude toward the study were included, three of whom had malignant melanomas of the choroid.

Seven eyes of seven patients were exposed to light for intervals of 30 to 45 minutes. Three eyes containing primary malignancies were enucleated $1^1/_2$ to $4^1/_2$ days after light exposure. Follow-up data on four blind eyes with clear media were gathered 15 to 36 months after light exposure. Studies before and after exposure included: best corrected visual acuity, clinical examination of the anterior and posterior segments, color fundus photography, fluorescein angiography, electro-oculography, and in eyes that were enucleated, ocular histologic study by both light and electron microscopy.

The studied eyes were fully dilated with 1% cyclopentolate HCl and 10% phenylephrine HCl. An indirect ophthalmoscope was used as a light source. The eyelids were held open with the examiner's fingers. With a setting of 7.5 V, light was focused on the retina through a condensing lens. Light was focused on the maculas in the four blind eyes with clear media. In the three eyes with tumors, light was focused on a predetermined area of the retina in the posterior polar region (Table 1). Because of the development of discomfort from the light effects, the corneas were anesthetized with repeated instillations of proparacaine. At

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TABLE 1

CLINICAL AND EXPERIMENTAL DATA ON THREE PATIENTS WITH CHOROIDAL MELANOMA

	Case 1	Case 2	Case 3
Age (yrs)	33	60	54
Melanoma cell type	Spindle A	Mixed	Mixed
Light exposure			
Duration (min)	45	30	45
Site	Retina nasal to disk	Macula	Retina inferior to disk
Time to enucleation (hrs)	42	38	113
Specimen preparation			
Time before immersion in 3% glutaraldehyde (min)	10	10	2
Interval between immersion and opening of eye (min)	10	30	Immediate

intervals of five to ten seconds, the corneas were bathed with artificial tears administered with an eye dropper, and rest periods of 30 seconds were allowed every five minutes. The total duration of light exposure was 45 minutes in each patient, except in one in whom the duration was 30 minutes (Case 2, Table 1).

The intensity of the light focused through the condensing lens was measured radiometrically and photometrically. This measurement determined the approximate light intensity at the cornea. Radiometric and photometric calculations were 0.42 W and 11,000 mL, respectively. Because of ocular discomfort associated with corneal erosion and edema that developed after light exposure in five patients, a second indirect ophthalmoscope fitted with a neutral density filter was used to reduce the amount of light delivered to the eyes of two patients (Cases 6 and 7). The intensity was measured at 0.005 W and 145 mL, indicating an effective filtration of approximately 99% of the light. The spectral distribution of light measured for the filtered and the nonfiltered ophthalmoscope was similar (Fig. 1). The luminance in millilamberts of the light sources used as they relate to

TABLE 2

CLINICAL AND EXPERIMENTAL DATA ON FOUR PATIENTS WITH UNILATERAL BLINDNESS

	Case 4	Case 5	Case 6	Case 7
Age (yrs)	22	29	46	70
Primary eye disease	Posttrauma orbital hematoma, optic nerve ischemia	Craniopharyngioma	Wegener's granulomatosis of orbit	Optic nerve meningioma
Light exposure to macula				
Duration (min)	45	45	45	45
Intensity	Maximal	Maximal	99% filtered	99% filtered
Evidence of retinal change				
Fluorescein angiography	No	No	No	No
Clinical appearance	No	No	No	No
Follow-up (yrs)	3	$2\frac{1}{2}$	$1\frac{1}{4}$	$2\frac{5}{6}$

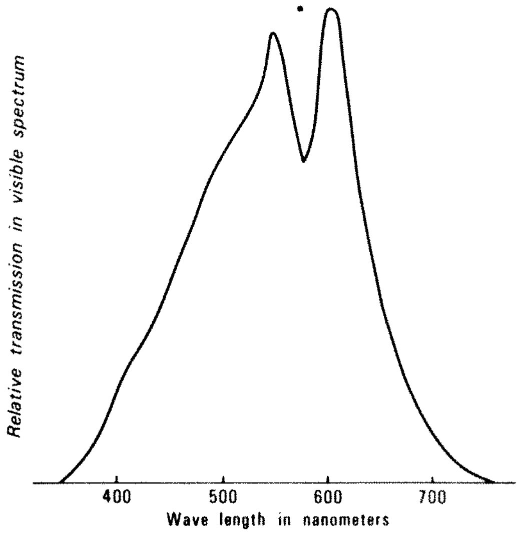

Fig. 1 (Robertson and Erickson). Spectral distribution measured from light of American Optical indirect ophthalmoscope through a 20-diopter Nikon condensing lens.

common visual sensations [14] is shown in Table 3.

At variable intervals after light exposure, the three eyes containing tumors were enucleated and immersed in glutaraldehyde between two and ten minutes after clamping of the optic nerve (Table 1). The eyes were all opened horizontally. Appropriate-sized tissue sections were taken from maximally exposed areas of the choroid and retina and from areas of the equatorial fundus remote from the site of the tumor and the site of maximal exposure. Tissue sections were processed by standard techniques for viewing with the electron microscope. In Case 3, small pieces of chorioretinal tissue were cut and separately fixed in glutaraldehyde 30 minutes after the initial fixation.

TABLE 3

LUMINANCE OF VARIOUS VISUAL SENSATIONS

Visual sensation	Millilamberts
Night vision	$\frac{1}{10,000}$*
Twilight	$\frac{1}{10}$ to 1*
Comfortable reading	10 to 100*
Snow in bright sun	8,000*
Light experiment	
Without filter	11,000
With filter	145

*Data from Ogle.[14]

Blind eyes exposed to light were evaluated within approximately one week of the light exposure and at variable intervals for a minimum of one to three years. The clinical appearance of the cornea, lens, retina, and choroid was recorded, and serial fluorescein angiograms were studied. In some instances, serial electro-oculography was done. In two patients, corneal thickness measurements were determined and corneal endothelial cell counts studied with the aid of specular endothelial photography.

RESULTS

In all patients receiving maximal exposure to light, the corneas were stripped of epithelium after approximately 20 minutes of exposure. This developed despite the 30-second rest periods and frequent irrigation with artificial tears. At the conclusion of the light exposure, all corneas had central stromal edema over an area 3 to 4 mm in diameter corresponding to the area of maximal light exposure. The corneal epithelium remained stripped centrally 24 hours later and, in some instances, extended to involve the entire cornea. Folds in Descemet's membrane were seen throughout the cornea, and the associated stromal edema was often diffuse (Fig. 2). In two eyes, cells and flare were observed in the anterior chamber. By 48 hours, the epithelial defects were generally covered, but stromal edema often persisted another 24 hours. In Case 3, stromal edema remained for more than four days.

Patients complained of discomfort after ten to 15 minutes of light exposure. Repeated topical instillations of anesthetics were then initiated to control the discom-

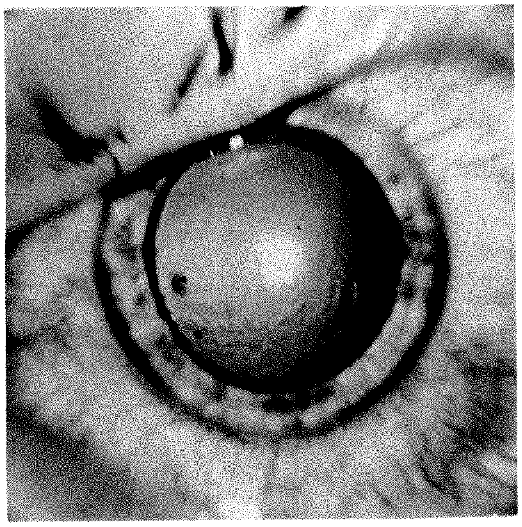

Fig. 2 (Robertson and Erickson). Case 2. Cornea 24 hours after light exposure. Stromal folds in Descemet's membrane are seen along with stromal edema and epithelial erosion.

fort. Several hours after light exposure, all patients who received maximal light exposure had intense ocular pain, which required the use of cycloplegics, eye patches, and codeine. Because of the corneal edema, ocular pain, and photophobia, studies scheduled to be repeated 24 hours after light exposure, such as dark adaptation, fluorescein angiography, and electro-oculography, were done with difficulty, delayed, or cancelled. In patients exposed to the filtered light, corneal damage was not observed either immediately or in the days after light exposure, and the patients had no ocular discomfort during or after exposure to light.

Results of dark adaptation studies in Case 1 were unchanged 24 hours after light exposure. In the three cases in which electro-oculography was performed both before and after light exposure (Cases 1, 5, and 7), no changes in the light peak-to-dark trough ratio were observed.

Visual acuities 24 hours after light exposure were diminished in all three patients with vision. However, we believed that the change in visual acuity in each patient was caused by the induced corneal edema rather than by a disturbance in retinal function. Visual acuity was diminished only from 6/15 (20/50) to 6/18 (20/60) in Case 1, from 6/6 (20/20) to 6/12 (20/40) in Case 2, and from 6/12 (20/40) to 6/30 (20/100) in Case 3, 24 hours after exposure.

In all three cases, the central corneal epithelium was absent. Corneal edema was not considered a significant pathologic finding. Central corneal endothelial cells studied by light microscopy were normal in all cases. In Cases 1 and 2, changes in the posterior pole seemed artifactual caused by delayed fixation.

Sections in Case 1 showed profound disruption of cell membranes in all layers of the retina. Inner and outer plexiform layers were so affected that only cellular debris was left in these areas. Photoreceptor cells showed destruction of inner and outer segments, with remaining structures exhibiting extensive disarray and varied degrees of membrane disruption. In Case 2, the retina was separated from the pigment epithelium in the area of intense light exposure. This separation was not observed in fundus photographs taken 24 hours after light exposure, at which time visual acuity was 6/12 (20/40), nor clinically during the minutes immediately preceding enucleation, and therefore the separation seemed artifactual. The pigment epithelium showed extensive disruption, with foci of intracellular and extracellular edema. Photoreceptor cells in the area of the detachment showed disrupted lamellae, thumbprint-like configurations in the inner end of the outer segments, and tubularization of outer segment lamellae.

Within two minutes of clamping the optic nerve, histologic changes seen in Case 3 were minimal and limited to the photoreceptor cells. Although findings on light microscopy were normal, abnormalities were seen with electron microscopy. Some lamellar membranes were disrupted, and irregular twisting and bending of

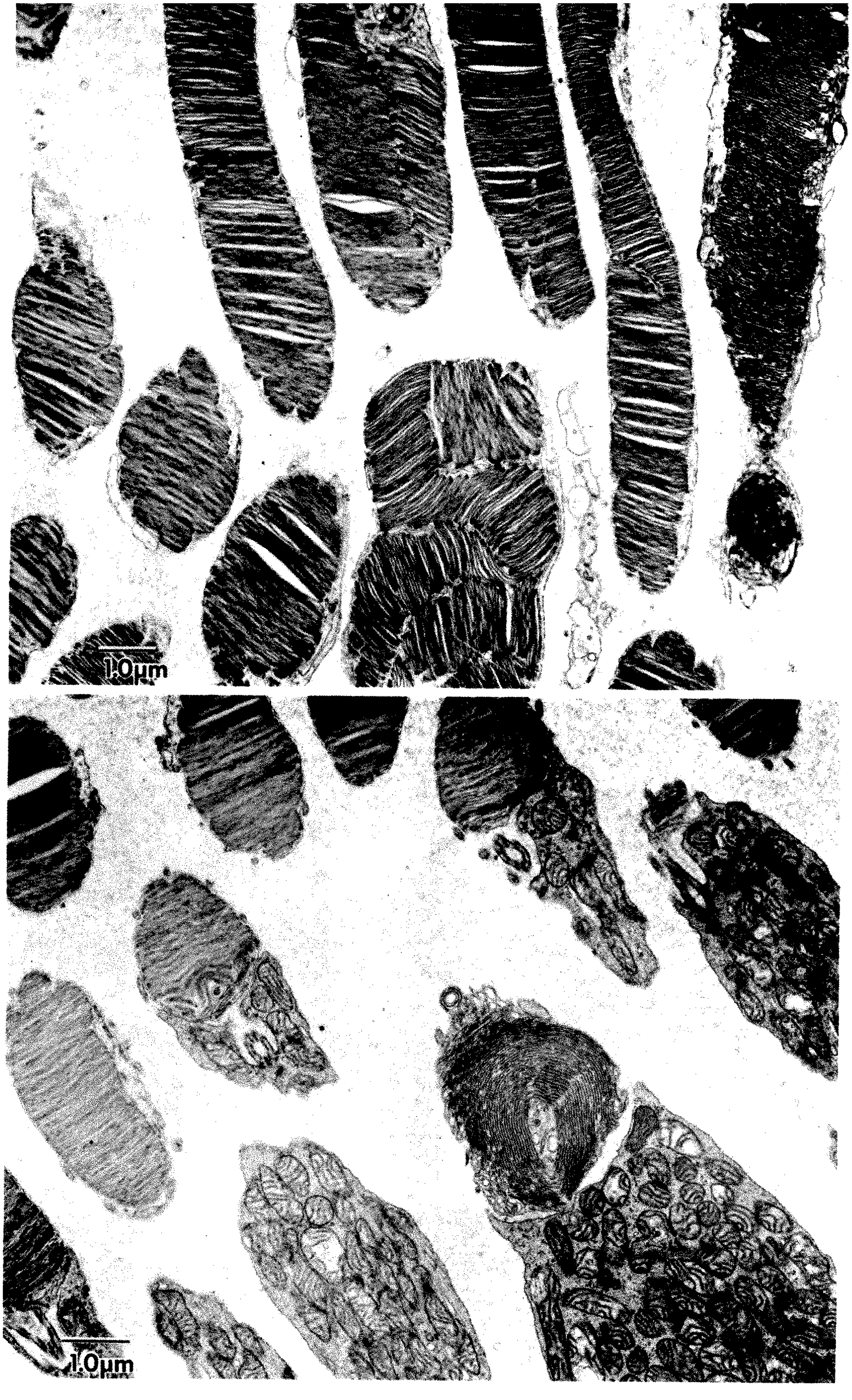
1.0µm
1.0µm

Fig. 3 (Robertson and Erickson). Case 3. Sections from sites of maximal light exposure. Top, Electron micrograph showing irregular twisting and bending of outer segments of retinal photoreceptors observed five days after light exposure (×6,000). Bottom, Irregular thumbprint-like configurations in inner ends of outer segments of retinal photoreceptors (×6,930).

←

the outer segments was seen occasionally (Fig. 3, top). Irregular thumbprint-like configurations in the inner end of the outer segments were observed in a few cells (Fig. 3, bottom). Some mitochondria of the inner segments of the photoreceptors were swollen. Although these changes were seen in sections of the equatorial retina, as well as in the site of maximal light exposure, all changes were seen with greater frequency in the area receiving the most intense light exposure. Pathologic changes were not noted in the inner retinal layers or in the pigment epithelium (Fig. 4).

Of the four blind eyes exposed to light, careful evaluation with slit-lamp biomicroscopy, ophthalmoscopy, color fundus photography, and fluorescein angiography failed to show any recognizable changes in the retina 15 months to three years after light exposure. Fundus photo-

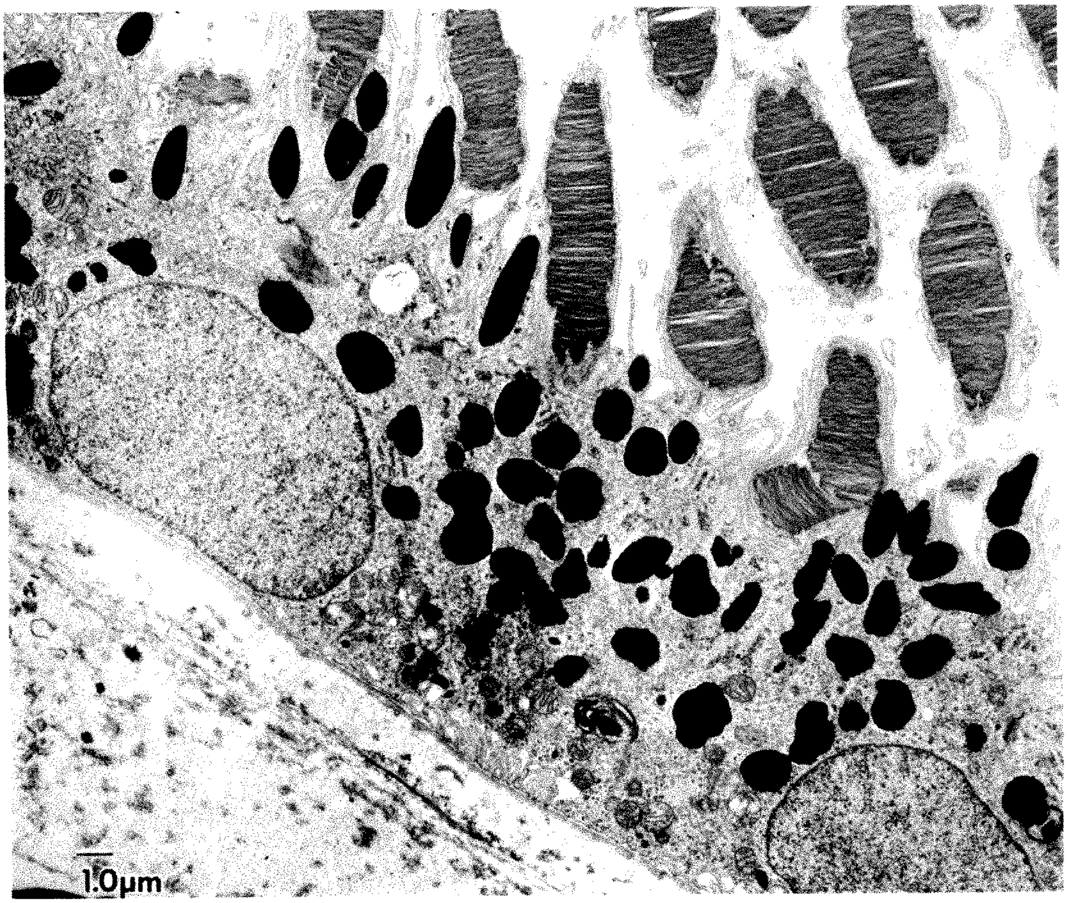

Fig. 4 (Robertson and Erickson). Case 3. Pigment epithelium and outer photoreceptor layer from area of retina remote from site of maximal light exposure. Note normal appearance of pigment granules in pigment epithelium and slight irregularity of lamellae of outer photoreceptors (×2,500).

graphs and fluorescein angiograms taken before and after light exposure in Case 4 are shown in Figures 5 and 6. Serial electro-oculography in Case 5 showed no change one week and 14 months after light exposure, when a comparison was made with the recording before light exposure.

Corneal thickness measurements and endothelial cell densities in Cases 4 and 5 determined 1½ to three years after exposure were considered to be normal. Although the cornea of the experimental eye in Case 4 developed peripheral changes similar to those seen in cornea farinata, the endothelial cells shown by specular photomicroscopy were normal (Fig. 7). Changes in the crystalline lenses were not observed in any of the experimental eyes.

DISCUSSION

The production of photic maculopathy in monkey eyes from the light source of the indirect ophthalmoscope was especially startling because photoreceptor damage and a visible pigmentary retinopathy could be induced after exposure intervals as short as 15 to 45 minutes.[9-11] The most significant effect that we observed in these experiments was the development of corneal edema. Despite our efforts to minimize drying by frequent irrigation during the periods of light exposure, we believe that the corneal edema occurred because of a drying effect from the heat of the light source; the use of topical proparacaine also may have contributed.

The observed changes in the retina of one adequately fixed eye enucleated four days after light exposure were limited to subtle changes in the photoreceptors visible by electron microscopy only. Although autolysis after enucleation could not be completely excluded as a cause of these subtle findings, lytic changes would be unlikely, because the tissue was appropriately fixed in glutaraldehyde within two minutes after the clamping of the optic nerve. However, if the observed changes were produced solely as a consequence of light exposure, experiments in subhuman primates reported by others suggest that these changes are reversible.[10] Abnormal findings in the other two enucleated eyes were probably a consequence of delayed fixation, allowing post-enucleation autolysis. Changes in visual acuity were explained on the basis of corneal changes.

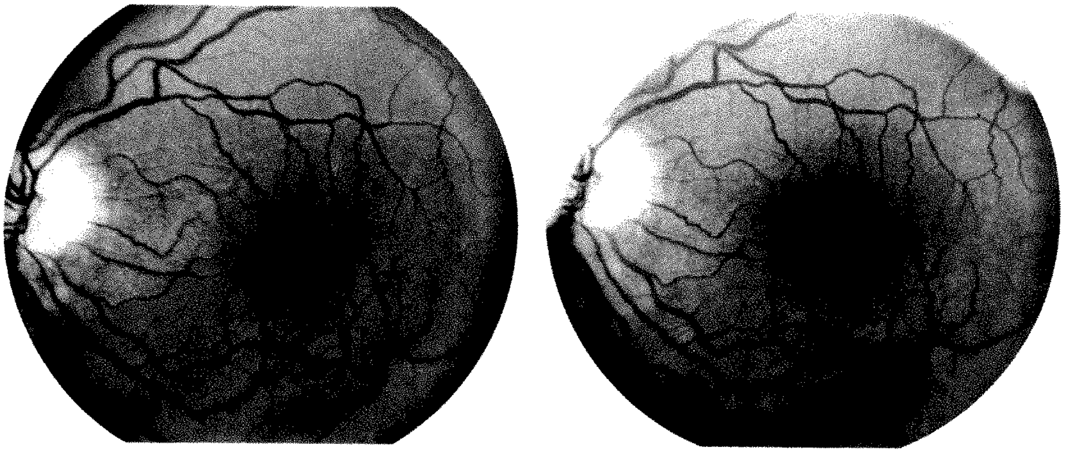

Fig. 5 (Robertson and Erickson). Case 4. Fundus before (left) and three years after (right) 45-minute exposure to maximal intensities of light delivered from indirect ophthalmoscope. No change is recognizable.

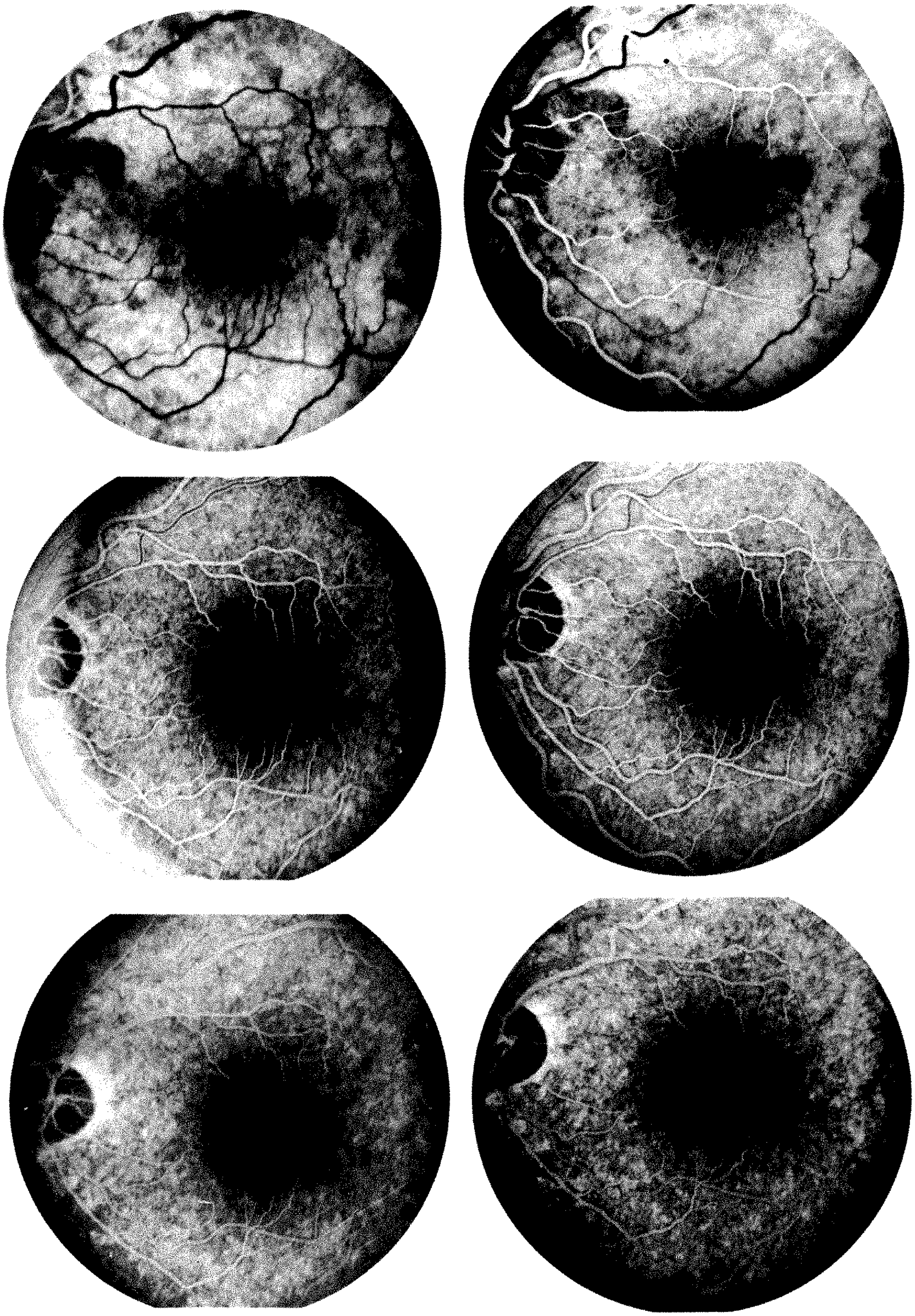

Fig. 6 (Robertson and Erickson). Case 4. Angiograms before (left) and three years after (right) 45-minute exposure to maximal intensities of light delivered from indirect ophthalmoscope. No change recognizable: choroidal flush (top left and right); arteriovenous phase (middle left and right); late phase (bottom left and right).

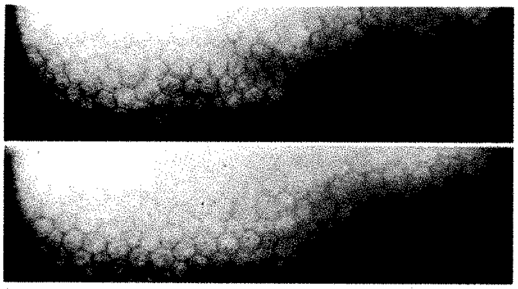

Fig. 7 (Robertson and Erickson). Case 4. Endothelial cells of corneas as seen with the clinical specular microscope. Top, Normal mosaic of central cornea of right eye. Bottom, Blind fellow eye three years after 45-minute exposure to maximal intensities of light from indirect ophthalmoscope. Endothelial cell counts and mosaic remain within normal limits.

The measured power of the light at the corneal surface was essentially the same as that calculated by Friedman and Kuwabara[8] in their monkey experiments. Because the light sources were the same in ours and the rhesus monkey experiments, differences in observed results cannot be the result of variations in the intensity of the more dangerous blue end of the spectrum.[15] Possibly, there is a true difference in susceptibility between human and monkey eyes. However, in Cases 4 to 7 the eyes were blind as a consequence of optic nerve damage and, therefore, the situation was not typical. However, the general appearance of the retinas was essentially normal, and such blind eyes can show photocoagulation burns and exhibit normal electroretinograms, so it cannot be assumed that these retinas would behave in a manner dissimilar from normal eyes. Possibly, a retinal effect was not observed in our human experiments because of the development of corneal edema. This may have caused enough light diffusion to diminish the intensity of focused light significantly on the target retina. The experimental conditions also differed from the subhuman primate experiments in that momentary slight deviations in the alignment of the light source and target retina occurred, and an allowance was made for 30-second rest periods every five minutes during the light exposure. Despite our inability to induce a photic retinopathy with indirect ophthalmoscopy, the human retina is susceptible to damage from high-energy light sources that are not used for retinal photocoagulation, as indicated by the unusual occurrence of retinal damage after observation of a welding arc,[16] and the development of retinal damage from intravitreal fiberoptic light.[17]

Although exposures to light from indirect ophthalmoscopy in routine clinical examinations are much shorter and less steady than those used in this experiment, even multiple consecutive prolonged meticulous retinal examinations rarely produce corneal edema as observed in this study. Our observations indicate that light exposures sufficient to cause this significant reversible anterior segment complication do not appear to be associated with significant retinal damage. We believe that photic retinal damage from routine clinical indirect ophthalmoscopy probably does not occur to any degree of clinical importance, because the much greater light exposure used in this experiment did not reduce visual (retinal) function and led to only subtle and probably reversible ultrastructural changes in the outer segments of the photoreceptors. However, it seems judicious in the clinical setting to delay further examination in the presence of developing corneal edema and ocular discomfort.

SUMMARY

In an experiment designed to simulate an excessively prolonged clinical examination, seven human eyes were exposed to light focused through a 20-diopter, aspheric condensing lens for 30 to 45 minutes. Three eyes contained primary malignant tumors of the choroid and were enucleated 1½ to 4½ days after light

exposure. Follow-up data on four blind eyes with clear media were evaluated 15 to 36 months after light exposure. Concentrated prolonged light exposure used in this experiment caused grossly visible progressive corneal edema, along with significant discomfort to the patient. Even with light exposures sufficient to cause these complications of the anterior segment, retinal injury was recognizable only with electron microscopy and was characterized by irregular bending and twisting of the outer segments of the photoreceptors, changes that are considered reversible in nonhuman primate studies. Intermediate to long-term follow-up of 15 to 36 months after light exposure to blind eyes failed to reveal lens changes or delayed light-induced degenerative changes in the retina recognizable by ophthalmoscopy, fundus biomicroscopy, or color and fluorescein fundus photography.

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VITREOUS WICK SYNDROME

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Endophthalmitis is a rare complication of cataract surgery. With the use of newer antibiotics and better diagnostic techniques the prognosis for useful vision has improved.[1] We describe herein the occurrence of endophthalmitis and its treatment in a patient with vitreous wick syndrome.

CASE REPORT

A 67 year-old-man underwent an uncomplicated intracapsular cataract extraction with a sector iridectomy in his left eye one month before admission here. A fornix-based flap was used. The patient was examined two weeks later by the surgeon and his eye was healing well. The cornea and anterior chamber were clear; the vitreous was clear and intact. Approximately three weeks after surgery, the patient had pain in the operated eye. Conjunctival injection, diffuse corneal epithelial edema, a hypopyon, and anterior vitreous haze were noted, and the patient was referred to one of us (B.D.S.) for further consultation and management.

At the time of initial examination visual acuity in the left eye was light perception with accurate projection. The cornea was mildly edematous, and displaced anteriorly by a yellow knuckle of vitreous protruding through the cataract wound at 11:30 o'clock. Intraocular pressure was 6 mm Hg and the anterior chamber contained a 1-mm hypopyon with 4+ white cells in the aqueous. A dense opacity was present in the anterior vitreous. This vitreous density extended in a wick-like fashion to the cataract wound at 11:30 o'clock and was apparent on the surface of the globe (Fig. 1). Anterior chamber and vitreous aspirates were obtained for culture and cytologic examination. A Giesma stain of the vitreous aspirate showed modest numbers of gram-positive cocci, which subsequently proved to be coagulase positive *Staphylococcus aureus*, sensitive to many antibiotics including methicillin sodium, oxacillin, and gentamicin.

The patient was given 6 g of methicillin and 120 mg of gentamicin daily in divided doses; 250 mg of probenecid was prescribed orally four times daily, and topical gentamicin drops were instilled hourly. Gentamicin (20 mg) was injected subconjunctivally every day for the first three days after admission. On the second day after admission, the patient received an intraocular injection of 0.3 mg of gentamicin through a limbal opening. The gentamicin was placed directly on the vitreous opacity. Within a few days the anterior chamber cleared, although the anterior vitreous condensation remained. One week after admission, the anterior chamber became shallow. An ultrasonogram revealed a large choroidal effusion, a bridging anterior vitreal membrane, and clear posterior vitreous (Fig. 2). An anterior vitrectomy was performed through a limbal opening with the O'Malley Ocutome the next day. The anterior vitreous condensation and the anterior cyclitic membrane were removed. The cataract wound dehiscence at the 11:30 o'clock meridian was cleaned and resutured. A culture of the vitreous removed during surgery grew *Pseudomonas*. However, because the eye progressively improved, it was decided that this growth was a contaminant and the patient continued to receive only topical gentamicin and methicillin IV. The antibiotics were discontinued on the second postoperative day. The cornea and vitreous remained clear and the anterior chamber was deep. The patient was discharged on the fourth postoperative day. Fourteen weeks after vitrectomy visual acuity was 6/15+ (20/50+), the anterior chamber was deep, and the cornea and vitreous were clear (Fig. 3).

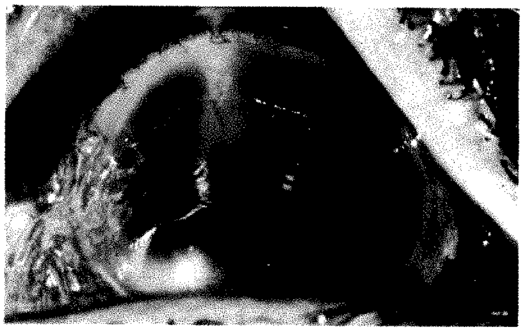

Fig. 1 (Srinivasan and associates). Left eye of patient at the time of admission. There is a haze in the central anterior vitreous with a wick extending to the cataract wound at the 11:30 o'clock meridian. Note exudate at 11:30 o'clock on the corneoscleral margin.

DISCUSSION

Ruiz and Teeters[2] described 11 patients with vitreous wick syndrome and divided these patients into three groups. The first

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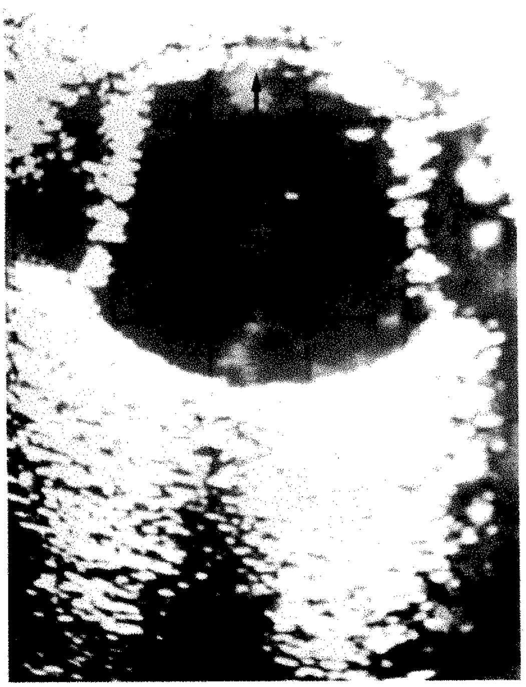

Fig. 2 (Srinivasan and associates). B-scan ultrasonogram of the left eye shows a large choroidal detachment (d), shallow anterior chamber, and a retroiridial bridging anterior cyclitic membrane (arrow).

group consisted of five patients in whom vitreous wicks developed, but no intraocular inflammation followed. The second group was composed of patients in whom vitreous wicks and intraocular inflammation developed. The third group was composed of two patients who had severe intraocular inflammation resulting in complete loss of vision, and in whom a diagnosis of vitreous wick syndrome was made retrospectively. Although laboratory confirmation of intraocular infection was not obtained in the four patients described in group 2, a hypopyon was present in one patient; in two patients yellow exudate extended along the vitreous strand from the fistulous opening in the cataract wound to the body of the vitreous. All patients in group 2 were given large doses of topical and systemic antibiotics as well as corticosteroids for several weeks until the inflammation subsided. Except for one patient in whom final visual acuity was hand motions caused by unrelated optic atrophy, the other patients successfully recovered visual acuity of 6/6 (20/20) to 6/9 (20/30). None of these patients had an anterior cyclitic membrane.

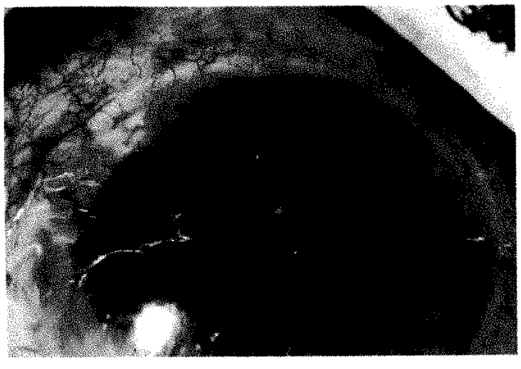

Fig. 3 (Srinivasan and associates). Left eye of patient four weeks after vitrectomy. The anterior chamber and vitreous are clear.

The vitreous wick syndrome produced a localized intraocular infection in our patient despite an onset of several days' duration. A partial explanation for the occurrence of localized infection may be that the strain of *S. aureus* involved was one of low virulence. Also, in experimental intraocular infection,[3] the anterior chamber had greater resistance than the vitreous to *S. aureus* organisms (inoculum size = 5,000 or less organisms), and the spread of infection from the anterior chamber to the vitreous was less rapid than from the vitreous to the anterior chamber. Finally, it is possible that the anterior portion of formed vitreous served as a barrier to confine the intraocular infection to the anterior segment.

Many investigators[4-7] have reported many advantages to performing a vitrectomy in cases of suspected infectious endophthalmitis. It may be used to secure biopsy material or to remove large infected areas of vitreous, and also to break posterior traction bands that may threaten retinal integrity.[4] Vitrectomy, in combination with intraocular antibiotic therapy, is gaining much support as an important treatment modality in endophthalmitis.[5-7] Cottingham and Forster[5] found that vitrectomy performed 24 hours after infection without intravitreal gentamicin did not cure *S. aureus* experimental endophthalmitis. They obtained better cure rates with a combination of intravitreal gentamicin and subtotal vitrectomy even when treatment was delayed 40 to 49 hours after onset of infection. Peyman, Vastine, and Raichand[6] prefer to do a vitrectomy 24 hours after the injection of intraocular gentamicin. In their series, patients who received intravitreal antibiotic therapy, with or without subsequent vitrectomy, within 36 hours of diagnosis had better visual acuity than patients in whom intravitreal antibiotic therapy was given later. Peyman performs vitrectomy as the primary procedure in bacterial endophthalmitis when the vitreous is severely involved and also in fungal endophthalmitis.

In our case, the anterior vitrectomy was performed at a time when signs of infection had abated but the hypotony and shallow anterior chamber remained unimproved. Ultrasonic demonstration of an anterior cyclitic membrane, and a large choroidal effusion suggested that the detachment of the ciliary body with resultant hypotony was caused by the cyclitic membrane. The purpose of the anterior vitrectomy was twofold: (1) to excise the cyclitic membrane to relieve the hypotony, and (2) to remove organized vitreous from the anterior aspect of the globe.

Thus, some patients who contract late postoperative intraocular infection secondary to a vitreous wick have a good chance for ocular preservation and restoration of useful vision. Prompt identification of the offending organism and adequate specific treatment, including intraocular antibiotics are essential for a good prognosis. A vitrectomy may be considered later in the course of the treatment, especially if vitreous membranes are present.

SUMMARY

A 67-year-old man had vitreous wick syndrome after an uncomplicated intracapsular cataract extraction with a sector iridectomy in the left eye. The intraocular infection cleared with antibiotic therapy including intravitreal antibiotic injection, but a residual anterior cyclitic membrane and shallow anterior chamber persisted. An anterior vitrectomy was performed later in the course of treatment after the intraocular infection subsided, to relieve the shallow anterior chamber, prolonged hypotony, and ciliary body detachment. Final visual acuity was 6/15+(20/50+) in the left eye.

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COMPARATIVE EVALUATION OF CURRENT OPHTHALMODYNAMOMETERS

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Ophthalmodynamometry, as a noninvasive evaluation of the pressure in the carotid artery system, has been available since Bailliart's original description in 1917.[1] The current techniques involve artificial increase in the intraocular pressure by compression or suction on the globe with simultaneous observation of the central retinal artery for the first complete pulsation for diastole and complete collapse for systole. These end points represent the pressures in the ophthalmic artery. Many investigators have described the usefulness of this test, but it is not used widely because angiography is more effective in detecting disease.[2-4]

Newer ophthalmodynamometers are now available that have a higher degree of accuracy and reliability.[5-7] We report herein a comparative study with the four commonly used ophthalmodynamometers to determine the variability between instruments and to establish the variation in normal individuals. The ophthalmodynamometers used were the Bailliart, Galin Digivac Suction Unit, Sisler Dynoptor, and the Stepanik Arteriotonograph.

SUBJECTS AND METHODS

Seventy-five volunteers, 31 men and 44 women, under 40 years of age with no history of symptoms of ocular, cardiovascular, or cerebrovascular disease were studied. The age limit of 40 years was chosen to minimize the possibility of undetected cardiovascular and cerebrovascular disease.

Each subject was placed in a sitting position for the duration of the test. Diastolic brachial artery blood pressure measurements were taken with the Roche automated ultrasound monitor and by routine auscultation in both arms.[8,9] If there was greater than a 3-mm difference between arms, the subject was excluded from the study.

The diastolic ophthalmic artery pressure was measured in the right and left eyes with each of the four instruments in exact accordance with operation instructions.[3,6,7,10] The order in which the instruments were used varied according to a predetermined randomization protocol. The diastolic end point was considered to be the first complete pulsation of the central retinal artery at the disk.

The time elapsed between the measurements with each instrument was sufficient to allow the intraocular pressure to return to the pretest state, usually from five to 15 minutes. The conversion from measured units to millimeters of mercury intraocular pressure was done with standard conversion curves provided in the operation manual of each instrument, except for the Stepanik unit where no conversion was necessary.[5,11] The coefficient of ocular rigidity was measured by using the Schiøtz tonometer with the 5.5, 10, and 15 g-weights and Friendenwald's 1955 nomogram.[12] The same observer made all measurements.

RESULTS

The age, sex, brachial artery pressure, intraocular pressure, ophthalmic artery pressures, and coefficient of ocular rigidity for each subject were recorded (Table 1). All pressures are given in millimeters of mercury. Because the Bailliart, Sisler, and Galin units do not read direct-

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TABLE 1

DIASTOLIC OPHTHALMIC ARTERY PRESSURES MEASURED WITH THE BAILLIART, SISLER DYNOPTER, GALIN DIGIVAC, AND THE STEPANIK ARTERIOTONOGRAPH ON 75 HEALTHY VOLUNTEERS UNDER AGE 40 YEARS*

Patient No., Sex, Age (yrs)	Brachial Pressure	Eye	IOP (mm Hg)	Ophthalmic Artery Pressure				Ocular Rigidity
				Bailliart	Sisler	Galin	Stepanik	
1,F,25	78	R	11	38	40	59	51	.0087
		L	12	38	41	55	53	.0128
2,F,36	76	R	19	37	51	52	32	.0160
		L	20	33	37	52	37	.0138
3,M,24	70	R	20	54	55	66	54	.0111
		L	21	55	57	65	56	.0107
4,F,32	88	R	22	69	71	65	60	.0121
		L	22	67	71	65	56	.0121
5,M,28	87	R	20	69	72	71	59	.0121
		L	19	66	72	73	56	.0121
6,F,23	83	R	20	47	65	61	67	.0163
		L	22	55	66	59	60	.0182
7,F,21	63	R	16	43	37	48	48	.0111
		L	18	43	39	46	48	.0106
8,M,30	74	R	19	50	45	50	46	.0100
		L	18	51	51	53	44	.0100
9,F,24	76	R	18	49	42	53	53	.0233
		L	20	53	52	52	45	.0228
10,F,31	73	R	19	56	44	54	56	.0186
		L	19	56	45	56	48	.0137
11,F,26	72	R	16	47	46	52	49	.0107
		L	16	50	47	57	48	.0118
12,F,21	69	R	18	42	49	51	36	.0134
		L	18	42	51	49	36	.0161
13,M,23	79	R	16	58	60	64	56	.0162
		L	16	54	67	61	55	.0146
14,M,27	80	R	17	46	49	55	56	.0094
		L	17	46	54	57	52	.0094
15,F,21	71	R	12	33	32	46	46	.0186
		L	13	39	40	42	45	.0186
16,M,28	72	R	16	52	52	64	55	.0190
		L	16	54	56	65	54	.0266
17,F,24	78	R	15	50	57	58	64	.0185
		L	15	54	52	62	62	.0185
18,F,29	84	R	17	56	60	57	56	.0134
		L	16	50	62	59	50	.0134

19,F,39	74	R	17	36	32	45	40	.0168
		L	15	34	33	42	36	.0168
20,F,28	71	R	17	49	46	57	44	.0185
		L	16	45	44	52	40	.0190
21,F,31	78	R	17	58	66	64	67	.0163
		L	16	55	63	64	65	.0190
22,F,20	79	R	18	53	63	60	60	.0106
		L	18	56	56	60	60	.0140
23,F,25	66	R	15	39	46	46	53	.0150
		L	14	37	46	45	52	.0166
24,M,27	74	R	18	55	56	57	52	.0122
		L	18	51	59	57	54	.0122
25,F,39	75	R	16	50	47	60	58	.0141
		L	16	51	51	60	52	.0126
26,M,27	76	R	12	43	40	54	50	.0126
		L	14	43	42	56	49	.0138
27,F,34	77	R	16	49	51	60	44	.0142
		L	17	51	54	59	46	.0170
28,F,18	60	R	18	39	39	44	43	.0149
		L	18	37	36	44	40	.0166
29,M,27	77	R	18	54	47	57	54	.0115
		L	17	53	54	57	54	.0115
30,M,18	66	R	14	39	43	58	50	.0126
		L	13	37	43	57	52	.0121
31,M,23	72	R	14	49	43	60	57	.0138
		L	14	50	49	61	58	.0138
32,M,24	73	R	15	50	46	60	52	.0123
		L	15	50	49	56	58	.0123
33,F,25	65	R	18	51	57	57	56	.0191
		L	18	51	59	57	56	.0190
34,M,22	82	R	17	57	62	62	66	.0119
		L	17	57	59	60	65	.0121
35,F,21	66	R	17	49	42	53	46	.0172
		L	17	49	44	49	45	.0153
36,F,20	71	R	18	45	51	53	49	.0282
		L	17	44	46	53	45	.0282
37,F,20	72	R	16	43	44	59	60	.0125
		L	16	47	50	65	56	.0125
38,M,26	81	R	18	53	59	62	58	.0146
		L	18	53	56	62	58	.0146
39,F,23	66	R	13	39	40	53	48	.0142
		L	13	38	41	51	44	.0155
40,F,24	66	R	14	36	37	55	44	.0137
		L	14	35	33	51	41	.0125

(Table continued on following pages)

TABLE 1 (*Continued*)

DIASTOLIC OPHTHALMIC ARTERY PRESSURES MEASURED WITH THE BAILLIART, SISLER DYNOPTER, GALIN DIGIVAC, AND THE STEPANIK ARTERIOTONOGRAPH ON 75 HEALTHY VOLUNTEERS UNDER AGE 40 YEARS*

Patient No., Sex, Age (yrs)	Brachial Pressure	Eye	IOP (mm Hg)	Ophthalmic Artery Pressure				Ocular Rigidity
				Bailliart	Sisler	Galin	Stepanik	
41,M,33	75	R	20	62	63	77	67	.0206
		L	19	59	59	74	68	.0236
42,M,18	76	R	13	48	52	66	54	.0150
		L	14	48	54	60	56	.0135
43,M,19	72	R	13	41	33	51	44	.0137
		L	14	39	34	51	46	.0160
44,M,25	78	R	17	68	69	75	64	.0170
		L	17	68	64	75	64	.0170
45,F,23	80	R	20	55	64	61	66	.0162
		L	19	53	65	63	66	.0184
46,F,21	76	R	16	40	47	52	47	.0141
		L	17	39	49	53	48	.0141
47,F,36	79	R	16	53	50	61	58	.0203
		L	15	51	55	63	60	.0203
48,F,18	67	R	18	47	43	53	52	.0216
		L	16	42	42	52	50	.0254
49,M,29	83	R	19	62	69	69	60	.0123
		L	19	66	70	68	68	.0107
50,M,22	79	R	16	43	50	59	54	.0190
		L	16	47	54	59	57	.0190
51,F,30	72	R	18	46	51	57	58	.0228
		L	18	47	51	53	56	.0273
52,F,19	64	R	16	40	43	48	46	.0140
		L	15	34	39	44	42	.0140
53,F,20	59	R	21	47	47	51	52	.0093
		L	22	44	53	51	50	.0128
54,F,27	81	R	15	47	55	56	61	.0239
		L	15	46	51	58	63	.0239
55,F,22	68	R	17	49	49	57	54	.0162
		L	17	49	49	53	56	.0162
56,F,25	81	R	16	47	47	64	52	.0163
		L	16	50	49	66	54	.0207
57,M,27	83	R	15	52	57	58	60	.0180
		L	15	49	55	63	58	.0180
58,M,24	74	R	15	46	52	57	52	.0146
		L	15	50	46	57	54	.0146

59,F,24	82	R	21	54	56	64	58	.0175
		L	22	51	55	63	58	.0199
60,F,32	79	R	18	54	47	60	57	.0126
		L	16	49	49	58	55	.0122
61,F,20	68	R	18	36	45	46	49	.0152
		L	17	35	37	41	46	.0152
62,F,18	71	R	18	56	50	57	57	.0190
		L	18	54	54	57	57	.0168
63,M,28	74	R	17	36	39	45	41	.0126
		L	16	35	40	40	40	.0209
64,F,28	77	R	17	52	55	62	56	.0190
		L	17	51	54	60	52	.0228
65,F,27	69	R	19	49	61	58	43	.0202
		L	19	45	53	58	47	.0202
66,M,40	73	R	17	54	60	70	61	.0152
		L	17	56	58	64	58	.0175
67,F,25	68	R	16	37	36	44	41	.0104
		L	16	39	40	48	42	.0104
68,M,22	83	R	14	42	43	55	54	.0173
		L	14	44	47	55	56	.0173
69,M,24	82	R	14	50	46	63	62	.0225
		L	14	50	48	61	64	.0264
70,F,23	74	R	13	49	52	57	61	.0161
		L	12	48	53	57	64	.0161
71,M,24	81	R	16	57	50	60	48	.0180
		L	15	55	53	59	52	.0134
72,M,25	83	R	18	49	48	60	53	.0193
		L	19	51	52	58	51	.0193
73,M,31	93	R	16	69	64	66	75	.0269
		L	16	71	69	71	74	.0172
74,M,29	74	R	12	46	43	57	55	.0196
		L	13	48	42	60	52	.0138
75,M,36	81	R	17	53	51	62	66	.0106
		L	17	52	54	62	56	.0111

*All pressures are given in mm Hg.

TABLE 2

MEAN AND STANDARD DEVIATIONS OF AGE, BRACHIAL PRESSURE, IOP, OPHTHALMIC ARTERY PRESSURE, AND OCULAR RIGIDITY IN 75 HEALTHY VOLUNTEERS UNDER AGE 40 YEARS*

				Ophthalmic Artery Pressure				
	Age (yrs)	Brachial Pressure	IOP (mm Hg)	Bailliart	Sisler	Galin	Stepanik	Ocular Rigidity
Mean	25.7	74.9	16.6	48.8	50.6	57.3	53.4	.0160
S.D.	5.3	6.6	2.3	8.2	9.2	7.2	8.0	.0043

*All pressures are given in mm of Hg.

ly in millimeters of mercury, their standard conversion curves were used.

The mean value and standard deviation for each factor in Table 1 was recorded (Table 2). The mean value and standard deviation for the ophthalmic artery pressure in all eyes expressed as a percentage of their respective brachial artery pressure are shown in Table 3.

The absolute values of the average percent difference between the right eye and the left eye are shown in Table 4.

Correlation coefficients for ophthalmic artery pressure and the ophthalmic artery to brachial artery pressure ratio were recorded for each combination of two instruments (Tables 5 and 6). The Stepanik unit measures the pressure directly, and was compared with each of the three indirect instruments to see if the pressure difference was correlated with coefficient of ocular rigidity. When the subjects were divided into two groups, one above and one below the mean rigidity of 0.0160, it was found that the group below this mean had a mean ophthalmic artery pressure with each indirect instrument that was from 1 to 2 mm Hg higher ($P<.01$).

DISCUSSION

The pressures obtained with these instruments are significantly different and partially explain the poor standardization of the test. The main cause for this difference is calibration. The Bailliart unit was calibrated by Magitot and Bailliart in 1919 using the Schiøtz tonometer, whereas the Stepanik unit uses a modification of the Mackay-Marg tonometer.[11] Both the Stepanik and Mackay-Marg have been shown to be within 2 mm Hg of the intraocular pressure measured with indwelling catheters on enucleated eyes.[7,13] Studies comparing the Mackay-Marg and Schiøtz tonometers indicate that the Schiøtz may read from 10 to 20% lower.[14,15] The Bailliart mean ophthalmic artery pressure is 9.0% lower than the Stepanik unit. To compensate for the Schiøtz

TABLE 3

MEAN AND STANDARD DEVIATION OF THE DIASTOLIC OPHTHALMIC ARTERY PRESSURE EXPRESSED AS A PERCENTAGE OF THE RESPECTIVE BRACHIAL ARTERY PRESSURE IN 75 HEALTHY VOLUNTEERS UNDER AGE 40 YEARS

	Bailliart	Sisler	Galin	Stepanik
Mean	65.0%	67.5%	76.6%	71.3%
S.D.	8.8%	10.1%	7.9%	8.9%

TABLE 4

THE MEAN AND STANDARD DEVIATION OF THE PERCENT RIGHT TO LEFT OPHTHALMIC ARTERY DIFFERENCE IN 75 HEALTHY VOLUNTEERS UNDER AGE 40 YEARS

	Bailliart	Sisler	Galin	Stepanik
Mean	4.5%	6.7%	3.6%	4.9%
S.D.	3.9%	5.7%	3.4%	4.1%

calibration error, ophthalmic artery pressures measured with the Bailliart unit would be more correct if values from the standard curves were increased by 10%.

Sisler adopted the Bailliart curve for use with his unit, but our study reveals that the Sisler unit measures approximately 4% higher than the Bailliart. His own pilot study showed it to be 14% higher.[16] We believe that the higher readings with the Sisler unit are the result of mechanical differences in the instruments. The Bailliart unit applies a 6.5-mm diameter convex surface to the temporal equatorial portion of the globe and displaces it against the nasal wall of the orbit. The Sisler unit, in contrast, applies an 8.8-mm concave surface to the cornea and displaces the globe against the apex of the orbit.

The variation in scleral thickness and fiber arrangement causes elasticity to vary throughout the globe; therefore ocular rigidity will vary with the point of applanation, resulting in a different IOP for a given force.[7] Because of these factors, significant differences exist.[17] Measurements with the Sisler unit should be increased from 5 to 6% over the values obtained from the Bailliart conversion curve.

All studies have shown that the suction technique yields from 7 to 12% higher readings when compared to the compression technique.[16,18,19] These higher readings are not a result of calibration but represent the different forces present in these techniques. In compression, a force equal to that exerted on the globe is transmitted to the orbital contents, and to some extent to the ophthalmic artery, resulting in a lower apparent central retinal artery pressure. Stepanik and Ossoinig[20] have shown 1- to 2-mm changes in the sagittal globe length during routine applanation tonometry. At the much higher pressures induced by ophthalmodynamometry, an alteration in the course of the ophthalmic artery would increase its resistance to flow. The resultant measured pressure in the central retinal artery would again be lowered. This transmitted orbital force is no problem provided it is consistent from patient to patient. Because there is no significant difference in the standard deviation in the Stepanik

TABLE 5

CORRELATION COEFFICIENT (r) FOR OPHTHALMIC ARTERY PRESSURE FOR EACH COMBINATION OF TWO INSTRUMENTS

Baillairt	1	—	—	—
Sisler	.83	1	—	—
Galin	.81	.76	1	—
Stepanik	.70	.67	.73	1
	Bailliart	Sisler	Galin	Stepanik

TABLE 6

CORRELATION COEFFICIENT (*r*) FOR OPHTHALMIC ARTERY TO BRACHIAL ARTERY RATIO FOR EACH COMBINATION OF TWO INSTRUMENTS

Bailliart	1	—	—	—
Sisler	.74	1	—	—
Galin	.69	.61	1	—
Stepanik	.57	.52	.61	1
	Bailliart	Sisler	Galin	Stepanik

unit and the Galin unit, the variability from compression forces on the ophthalmic artery is not significant. We believe that the 7 to 12% lower readings by compression result from these forces on the ophthalmic artery.

That each of the indirect instruments has its lowest correlation with the Stepanik unit implies that the variables with indirect instruments are similar; the Bailliart and Sisler units have the highest correlation of pressure measurements, reflecting the similarity of the two compression devices.

The coefficient of ocular rigidity represents a small (1 to 2 mm Hg) but statistically significant factor in the difference of the mean values measured with the direct and the indirect units ($P<.01$). For an individual patient, however, it is of no predictive value in determining the error introduced by using the standard conversion curve. The coefficient of rigidity is known to be a function of the measuring apparatus, the intraocular pressure, and the perfusion pressure.[21,22] Because these parameters change during ophthalmodynametric measurements it is not surprising that smaller studies could show no statistical significance in the ocular rigidity as a cause for error with indirect instruments.[18]

With each instrument, the standard deviation of the ophthalmic artery to brachial artery pressure ratio was greater than the standard deviation of the ophthalmic artery pressure alone. The ophthalmic artery pressure is dependent on many factors, such as the exact caliber of the ophthalmic artery, the total vascular resistance distal to the ophthalmic artery, the angle of branching, and the number of ophthalmic artery branches. These factors result in a 30% variability in this ophthalmic artery to brachial artery ratio, for a 95% confidence level.

We cannot assume that the intraocular pressure is equal to the central retinal artery pressure upon collapse, unless the central retinal artery rigidity is negligible. In young individuals, as in our study, this is probably not an important factor. However, with advancing age various amounts of arteriolar sclerosis will increase the rigidity of the small vessel wall, resulting in falsely high readings such as those that have been reported higher than the systemic pressure. The presence of disease in the carotid artery tending to lower the ophthalmic artery pressure is obscured by increased rigidity in the abnormal, small vessel. Being able to determine the small vessel rigidity effect and correcting for it is valuable. It will not only reduce the variability in ophthalmic artery measurement, but it will also yield an index of small vessel disease.

Each of the instruments yields a mean right to left pressure difference of approximately 5%. The Galin unit was the smallest at 3.6% and the Sisler unit the largest at 6.7%. This small difference with all instruments reflects the near symmetry of the vascular system and provides a useful index in evaluating carotid disease. Our study indicates that in normal individuals there should be no difference greater than 8 to 10% between eyes ($P<.05$).

Carotid disease is often bilateral resulting in many cases of severe obstruction with little asymmetry in pressure measurements.[23] The large variability in the absolute ophthalmic artery pressure and the frequency of bilateral carotid disease would cause a significant number of diseased individuals to fall within our normal limits.

Use of standard conversion curves can lead to large errors in the apparent pressure (Table 1). To avoid this problem, the force necessary to achieve systolic and diastolic end points should be recorded. Then this same force is reapplied and the induced intraocular pressure measured by Goldmann applanation tonometry.

Limits of ± 15% for the ophthalmic artery to brachial artery pressure ratio and 10% for right to left pressure difference should be used for 95% confidence levels. The normal mean ophthalmic artery pressure is dependent on age and should be standardized along with correction factors for arteriolar rigidity to make this procedure more accurate.

SUMMARY

Seventy-five patients under 40 years of age with no history of ocular, cardiovascular, or cerebrovascular disease were examined with four commonly used ophthalmodynamometers. There was a significant difference in the absolute values measured with each instrument and the ophthalmic to brachial artery pressure ratio. The standard deviations were comparable between 8.1 and 10.2%.

The absolute value of the average percent difference between the right and the left eye ranged from 3.6% with the Galin unit to 6.7% with the Sisler unit. Limits of ± 15% for ophthalmic artery to brachial artery pressure ratio and 10% for right to left pressure difference should be used for 95% confidence levels. There was a small but definite correlation between the ocular coefficient of rigidity and pressure differences between direct and indirect instruments.

ACKNOWLEDGMENTS

Berkeley Bio-Engineering International, Inc.; Coburn Optical Industries, Inc.; and American Optical, Inc., provided the Stepanik, Galin, and Sisler ophthalmodynamometers, respectively.

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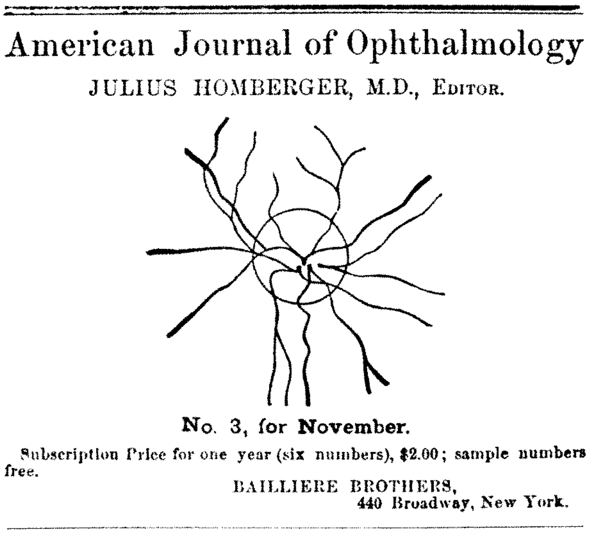
OPHTHALMIC MINIATURE



American Journal of Ophthalmology

JULIUS HOMBERGER, M.D., Editor.

No. 3, for November.

Subscription Price for one year (six numbers), $2.00; sample numbers free.

BAILLIERE BROTHERS,
440 Broadway, New York.

Am. Med. Times Dec. 18, 1862

POSTLENTICULAR MEMBRANE ASSOCIATED WITH SMITH-LEMLI-OPITZ SYNDROME

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Smith, Lemli, and Optiz[1] described an association of multiple congenital anomalies in three unrelated boys and more than 50 such case reports have since been described. We describe herein a boy with Smith-Lemli-Opitz syndrome who developed bilateral cataracts, posterior synechiae, and a dense postlenticular membrane that was noted at surgery. To our knowledge, this is the first published description of an associated postlenticular membrane and only the second with posterior synechiae.[2]

CASE REPORT

A 6-day-old boy was first seen by us in December 1972. He had multiple congenital anomalies including microencephaly, micrognathia, short nose, large nasolabial distance, low-set ears, epicanthal folds, cleft palate, syndactyly of second and third toes of both feet, polydactyly of the right hand and foot, ambiguous genitalia with complete bifid scrotum, severe hypospadius with minute penis, pyloric stenosis, congenital hip dysplasia, and severe psychomotor and growth retardation. These findings were consistent with the diagnosis of Smith-Lemli-Optiz syndrome. The chromosomal pattern revealed 46 chromosomes with normal male characteristics. Initial ocular examination revealed blepharophimosis in each eye without evidence of microphthalmia. The lenses were described as clear. Dilated examination was unremarkable (Fig. 1).

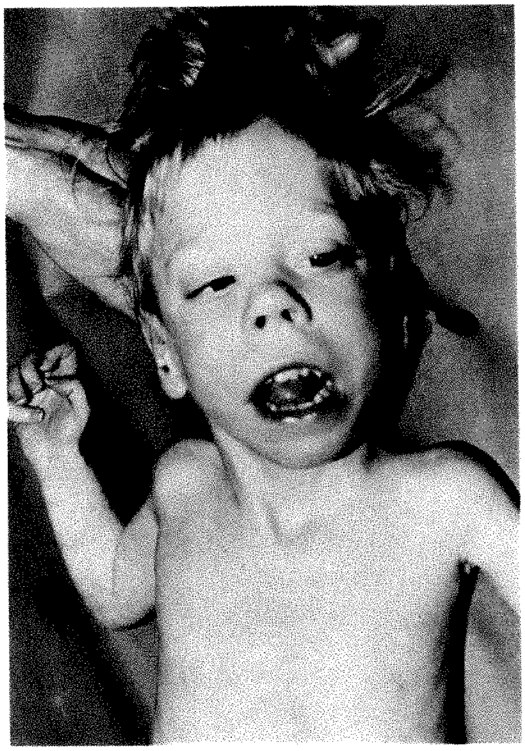

Fig. 1 (Freedman and Baum). Present case of Smith-Lemli-Opitz syndrome in a 5-year-old patient.

In February 1973 another dilated examination revealed no abnormalities of the lens, vitreous, or fundus. The child was seen next in March 1975 because the mother had noted white pupils over a three-month duration and a decrease in environmental awareness without photophobia. On ocular examination, the child did not respond to strong light stimulation. Bilateral blepharoptosis, worse in the right eye, was noted. The anterior segment was otherwise unremarkable except for posterior synechiae in each eye. The pupils were 3 mm in each eye and reacted briskly and equally. Intensive dilatation failed to break any synechiae but did reveal dense cataractous lenses in each eye. The vitreous and fundus could not be visualized in either eye.

In June 1975 an aspiration of the cataract (Fig. 2) of the right eye was performed to increase the child's visual contact. At surgery, a dense white fibrous postlenticular membrane was noted. A vertical incision through the center of the membrane created an oval window (Fig. 3) that allowed a view of the vitreous and fundus. On postoperative examination the patient followed light occasionally. The right optic disk appeared small and pale. A pattern and flash visual-evoked potential after correction revealed a normal response in the right eye. No signals were present even to flicker in the left eye, probably because of the density of the cataract. The patient died in May 1977. No autopsy was performed.

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AMERICAN JOURNAL OF OPHTHALMOLOGY 87:675-677, 1979

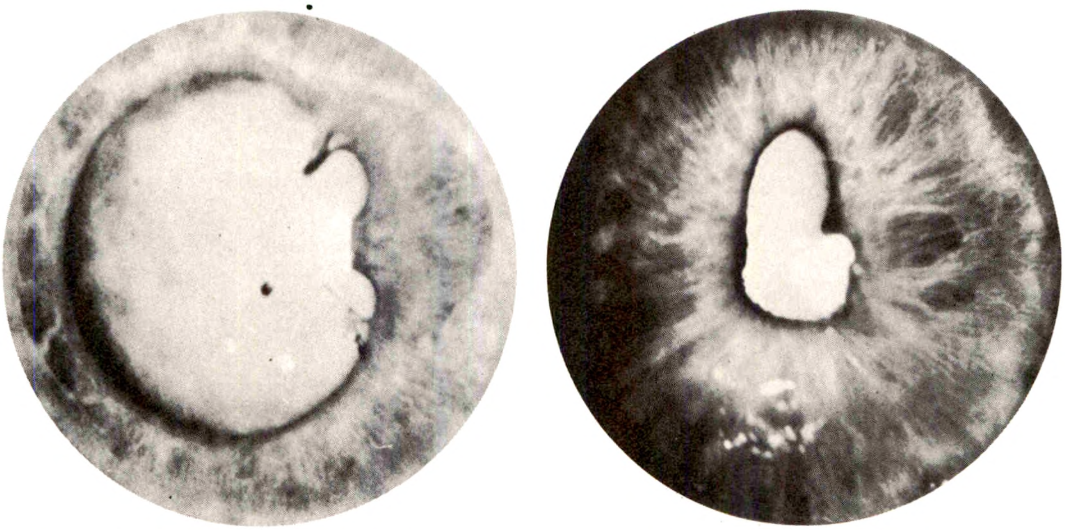

Fig. 2 (Freedman and Baum). Left, Cataract, right eye, pupil dilated. Right, Cataract, left eye, pupil undilated. Note posterior synechiae in each eye.

DISCUSSION

The Smith-Lemli-Opitz syndrome characteristically affects multiple organ systems including the eyes. Major systemic abnormalities include growth deficiency, moderate to severe mental retardation, microcephaly, short nose with anteverted nares, broad maxillary alveolar ridge, polydactyly and syndactyly of the second and third toes, hypospadias, cryptorchidism, pyloric stenosis, and whorls of the finger tips. Many other associated findings have been reported. Minimal diagnostic criteria for the Smith-Lemli-Opitz syndrome include anteverted nostrils, syndactyly of the second and third toes, and a male genital anomaly in a mentally retarded patient of short stature. The syndrome appears to follow an autosomal recessive pattern of inheritance.

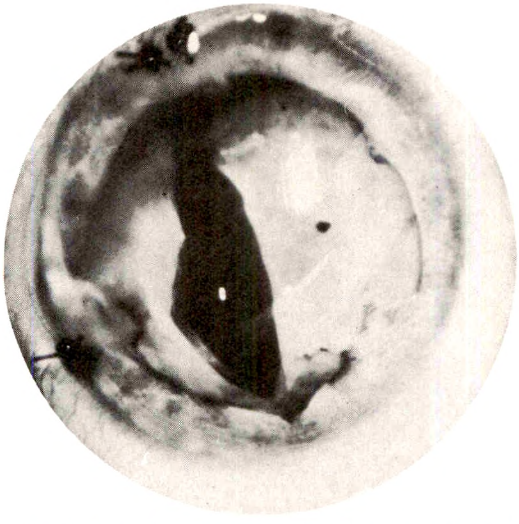

Fig. 3 (Freedman and Baum). Dense fibrous postlenticular membrane after incision, right eye.

Multiple ocular abnormalities are associated with this syndrome. In the original description[1] blepharoptosis, epicanthal folds, and strabismus were reported. Opitz and associates[3] subsequently added the following features: small palpebral fissures with antimongoloid slant, mild exophthalmos, retinal pigment epithelial defects, pale disks, nystagmus, microcornea, and aniridia. Duane's retraction syndrome, absence of lacrimal puncta, corneal enlargement, iris deformity, and optic pits were described by Gold and Pfaffenbach.[4] Cataracts associated with the Smith-Lemli-Opitz syndrome were first reported in 1968.[5] Opitz and associates[3] later described six cases of congenital cataracts associated with the syndrome. The first case of Smith-Lemli-Opitz with cataracts and posterior synechiae was reported by Cottlier and Rice.[2] In their patient, cataracts were first noted at age 10 months and were characterized as bilateral anterior cortical opacities. Posterior synechiae were also noted. The patient underwent an uneventful extracapsular cataract extraction. Sclerocornea associated with the Smith-Lemli-Opitz syndrome was described by Harbin and associates.[6]

SUMMARY

A 6-day-old boy with Smith-Lemli-Opitz syndrome developed bilateral cataracts, posterior synechiae, and a dense postlenticular membrane. No other signs of inflammation were noted that could account for the development of either the posterior synechiae or membrane. To our knowledge, this is the first published report of such an associated membrane.

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IMMUNE COMPLEXES IN UVEITIS

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Although various abnormalities have been observed in human uveitis, it is unclear whether they are important in the pathophysiology of these diseases or whether they are epiphenomena secondary to nonimmunologically mediated tissue damage.[1] Immune complexes consist of antigen and antibody and can result in immunologically mediated tissue injury. Circulating immune complexes have been demonstrated to cause ocular tissue destruction in experimental models of uveitis.[2] In retinal vasculitis and Fuchs' heterochromic cyclitis, immune complexes have been demonstrated in patients' sera; however the nature of these complexes and their correlation with disease status has not been delineated.[3-4]

We describe herein the results of our study demonstrating the existence of antigen-antibody complexes in the circulation of patients with chronic idiopathic iridocyclitis and diffuse uveitis. We have partially characterized these immune complexes and correlated the increased levels observed with disease activity.

SUBJECTS AND METHODS

We studied all patients with chronic iridocyclitis or chronic diffuse uveitis examined at the Uveitis Survey Clinic here, from 1977 to 1978 who did not have any detectable systemic disease. Fourteen patients with idiopathic chronic diffuse uveitis ranged in age from 9 to 73 years, with a median age of 44 years. Nine were women, five were men; 12 were white, one was black, and one was Oriental: The 16 patients with chronic idiopathic iridocyclitis ranged in age from 5 to 70 years. Fifteen were white and one was black. Twelve were women and four were men. Most patients were not receiving systemic corticosteroids; those who were had received their last dose at least 24 hours before testing. After informed consent was obtained, 20 ml of peripheral venous blood was drawn. The serum samples were coded and stored at −70°C. The severity of inflammatory disease in each patient was graded by summing numeric values assigned to the various clinical factors of uveitis[5]: degree of anterior chamber cell and flare (0 to 4), presence of keratic precipitates (0 or 1), degree of vitreous reaction (0 to 4), and presence of retinal vascular permeability as manifested by optic nerve or macular edema (0 or 4). This sum score for each patient (and the various clinical factors on which it was based) were separately compared to patient rank order on the basis of immune complex levels by using nonparametric Spearman coefficient analysis.[6]

Serum samples were tested in triplicate to determine the level of immune complexes using the Raji cell radioimmunoassay. This assay is a modification of a technique originally described by Theofilopoulos, Wilson, and Dixon,[7] and measures the uptake of complement fixing immune complexes by the C3 receptors on a human lymphoblastoid cell line. We diluted 0.025 ml of serum 1:4 with normal saline and incubated for 45 minutes at 37°C with Raji cells and rabbit antihuman immunoglobulin labeled with iodine 125, plus 1% human serum albumin. The Raji cells were then exten-

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From the Department of Ophthalmology and the Francis I. Proctor Foundation, University of California(San Francisco. This work was supported in part by National Institutes of Health grants EY01441, EY02072, and EY01759. Dr. Char is a recipient of Research Career Development Award 1K04EY00117.

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sively rewashed and the cell pellet counted in the gamma counter. Each patient's test results were compared with a standard curve in which known amounts of aggregated human immunoglobulin plus complement were incubated under similar conditions with the Raji cells. The levels of immune complexes were expressed in microgram equivalents of aggregated human immunoglobulin per milliliter. In most cases the replicates varied by less than 5%. In previous work from this laboratory, approximately 100 normal volunteers, ten patients after cataract surgery, and ten patients with chronic cyclitis had immune complex levels of 10±5 μg equivalents of aggregated human immunoglobulin per milliliter. We therefore set the upper limits of normal at less than 20 μg equivalents of aggregated human immunoglobulin per milliliter (± 2 S.D.).[8]

Increased levels of immune complexes detected by the Raji cell assay were confirmed by Clq binding and by precipitation of the immune complexes with 3% polyethylene glycol.[9,10] Immune complexes separated from the serum by 3% polyethylene glycol precipitation were further isolated by a combination of gel and affinity chromotography using Ultragel AcA-22 and Protein A-Sepharose CL-4B.[11] The molecular weight of the isolated immune complexes was determined by molecular sieve chromotography with Sepharose 4B. We used immunoelectrophoresis to characterize further the purified immune complexes.

RESULTS

Ten of 16 patients with chronic iridocyclitis showed increased levels of immune complexes (Fig. 1). The increases in immune complexes detected by the Raji cell assay showed a statistically significant correlation with uveitis disease activity ($r=0.56$; $P<.05$). Comparison of rank order on the basis of anterior chamber reaction vs immune complex increase also demonstrated a significant correlation ($P<.05$).

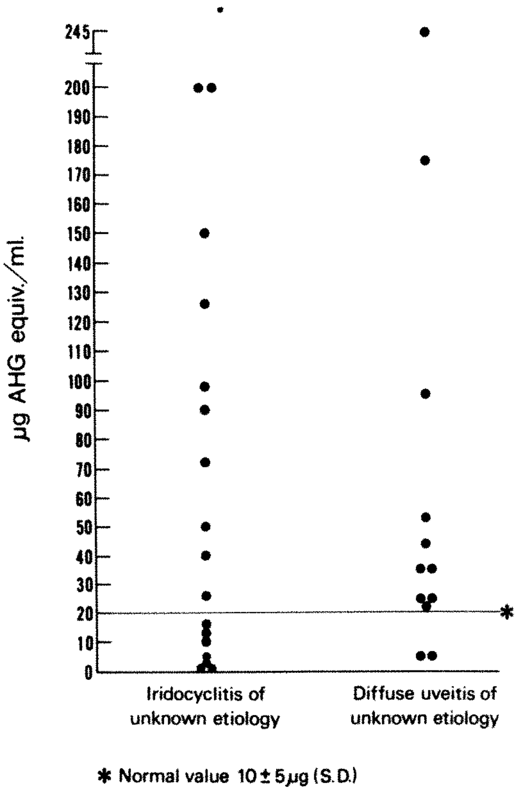

Fig. 1 (Char and associates). Immune complexes in uveitis.

Ten of 12 patients with chronic diffuse uveitis had increased levels of immune complexes; however, there was no significant correlation between immune complex levels and total disease activity, anterior chamber reaction, or vitreous reaction ($r=0.21$).

Confirmation of the increased levels of immune complexes in serum samples, as measured by the Raji cell radioimmunoassay, was obtained by Clq binding and by precipitation of the immune complexes in 3% polyethylene glycol (unpublished date). Although there was some interassay variation, the increased immune complex levels were confirmed for all patients.

Immunoelectrophoresis of the partially purified complexes demonstrated the presence of antibody of IgG type in the high molecular weight peak (Fig. 2). Immune complexes isolated from the sera of six patients had molecular weights between 2 and 4 × 10^6 daltons. When this material was eluted from Protein-A Sepharose affinity chromotography columns and tested by the Raji cell radioimmunoassay, significant activity confirmed the presence of immune complexes (unpublished data). A representative fractionation is shown in Figure 2.

DISCUSSION

Immune complex levels have correlated with both disease activity and prognosis in many nonocular human diseases; evidence for a similar correlation has been found in patients with the neuro-ophthalmic form of Behçet's syndrome.[12-19] In our study, ten of 12 patients with diffuse uveitis and ten of 16 patients with chronic iridocyclitis had significant increases in immune complex levels. In the latter group a significant correlation was established between these increased immune complex levels and disease activity.

Immune complexes may be important in the pathophysiology of human ocular inflammation; however, it is unclear whether they are a primary cause responsible for the development of the disease, or are merely secondary to nonimmunologic tissue destruction. In experimental models of lens-induced uveitis and retinal vasculitis, pathologic changes consistent with immune complex formation have been described.[20,21] Further, increased vascular permeability, an essential component of chronic experimental uveitis, can be produced experimentally by the intravenous injection of immune complexes.[22] Nonspecific fixation of immune complexes of any type conceivably could produce alterations in uveal permeability with the resultant uveitis. Our findings and those in previous work on immune complex levels in ocular inflammation are consistent with the hypothesis that immune complexes may be important in these diseases. However, further work must be done to clarify their role.

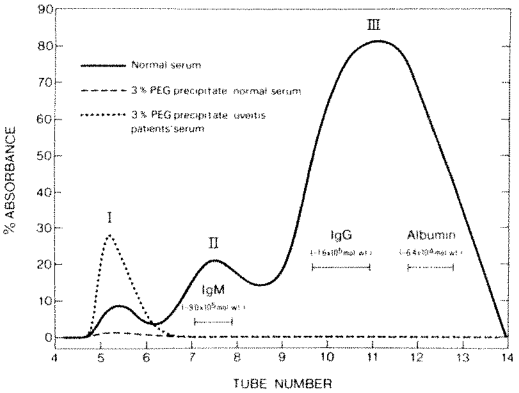

Fig. 2 (Char and associates). Ultragel AcA-22 chromatography of polyethylene glycol precipitate elution profiles.

The nature of the immune complexes detected in our study is unclear. Their molecular weight, estimated by column chromatography was approximately 2 × 10^6 daltons, which is similar to that reported in many other human diseases.[15,16,19,23-25] IgG was the predominant immunoglobulin present in these complement-fixing immune complexes. The antigenic component of these complexes is unlikely to be either viral or bacterial.[26] Rather, it is probably an ocular tissue surface antigen, an extraneous extraocular antigen, or an altered immunoglobulin. Although most uveitis patients do not have detectable autoantibodies, an anti-idiotypic antibody may have been responsible for the development of these immune complexes.

Because the morbidity associated with uveal biopsy is still too great to allow for the direct measurement of immune complexes in vivo, their role in the pathophysiology of uveitis must be determined in vitro. Conceivably, further studies of immune complexes may allow us to increase our diagnostic and prognostic accuracy in patients with these diseases and to develop more rational methods of therapy.

SUMMARY

We determined the incidence of immune complex increases, the nature of immune complexes, and the correlation between the immune complex level and clinical uveitis status in patients with chronic iridocyclitis and chronic diffuse uveitis. Ten of 16 patients with chronic iridocyclitis had increased levels of immune complexes, and this correlated with disease activity. Immune complexes were detected in ten of 12 patients with chronic diffuse uveitis. These complexes have a molecular weight of approximately 2×10^6 daltons. The antibody component of immune complex is IgG; however; the antigenic component of the immune complexes is unclear.

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CRYPTOCOCCAL MENINGITIS AND INTERNAL OPHTHALMOPLEGIA

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Almost half of the patients with cryptococcal meningitis have abnormal eye signs. Papilledema is the most common sign and blurred vision is the most common symptom. We describe herein a patient with unique ocular findings of bilateral internal ophthalmoplegia, bilateral sixth nerve paresis, and papilledema secondary to central nervous system cryptococcosis.

CASE REPORT

In May 1975, a 17-year-old girl developed a malar rash, alopecia, and arthralgias. Her antinuclear factor titer was 1 to 512, DNA binding capacity was 85% (normal, less than 20%), and a lupus erythematosus preparation was positive. A renal biopsy showed diffuse proliferative lupus nephritis. Systemic lupus erythematosus was diagnosed. She was treated with 20 to 40 mg of prednisone per day and 100 to 150 mg of azathioprine per day.

During the next 18 months, she was well, but then developed a fever up to 40°C. She was admitted here where a study for hidden infection was negative. Examination of her urine, cerebrospinal fluid, and blood included Gram stain as well as cultures for bacteria.

Further negative results were obtained in urine for cytomegalic inclusion disease, throat cultures including study for Epstein-Barr virus, and rectal swab for *Candida*.[1]

About one month later, she became irritable. She had rage reactions, nightmares, and heard voices. Her temperature ranged up to about 40°C. All previous tests results were again negative. Opening pressure on spinal tap was normal and the fluid was clear. There were no cells; protein and glucose were normal.

Prednisone was decreased from 60 to 30 mg per day. With this change in therapy, the patient became more alert and oriented. One month later she suddenly noted the onset of horizontal diplopia and blurred vision associated with her daily fever spikes. Her past medical history was noncontributory.

The patient was febrile, cushingoid, lethargic, and had a malar rash. She was somewhat confused, although she was no longer having hallucinations. She had a mild tremor of both hands, finger to nose ataxia, and a broad-based gait.

On neuro-ophthalmologic examination, visual acuity at distance was 6/6 (20/20) in both eyes. Near visual acuity was 6/60 (20/200) in both eyes. Both pupils were 6 mm and reacted sluggishly to light and near (Figure). Her amplitude of accommodation was less than 1 diopter in both eyes.

She had bilateral sixth nerve palsies with an inability to abduct either eye 20 degrees beyond midline. There was no blepharoptosis or other limitation of ocular movement. Cranial nerves were normal. Slit-lamp examination was normal. Tensions were 18 mm Hg in both eyes. Visual fields showed enlarged blind spots. There was bilateral optic disk edema. The vitreous was clear. The vessels, macula, retina, and periphery were otherwise normal.

An electroencephalgram was normal. Computed axial tomography showed mild increase in the size of the ventricles. Spinal fluid cultures were taken again. A lumbar puncture had an opening pressure of 330 mm Hg with 46 white blood cells per high powered field. The protein was 115 mg/100 ml and glucose was 17 mg/100 ml. India ink preparations were positive for encapsulated yeast and cultures subsequently identified *Cryptococcus*. The patient was treated with intravenous amphotericin on a daily basis for 21 days and then on alternative days for 21 days.

During the next month she gradually improved. About 14 months after the onset of her illness, her

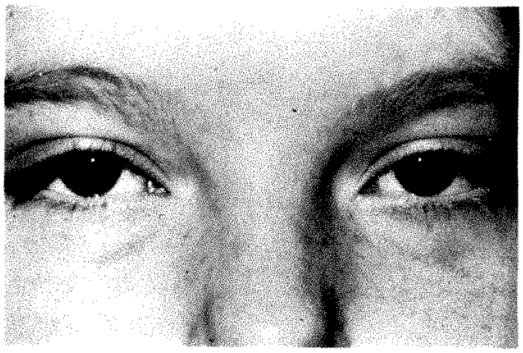

Figure (Lesser and associates). Patient with internal ophthalmoplegia.

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visual acuity was 6/6 (20/20) at distance and near. Accommodation was 8 diopters in each eye. Both pupils were 3 mm and reacted briskly to light and near. The papilledema and sixth nerve palsies had cleared. The patient was alert and oriented.

DISCUSSION

Although initial signs and symptoms may be present in lungs, lymph nodes, or bones, cryptococcosis has a predilection for neural tissue. Cryptococcosis is the most common mycotic infection of the central nervous system.[2]

About 70% of patients have neurologic symptoms, and autopsy discloses central nervous system involvement in 90%. Central nervous system involvement is usually secondary to hematogenous spread from a pulmonary lesion.[3] About 50% of affected patients have some underlying debilitating disease such as lymphoma or leukemia.[4]

Patients may have signs and symptoms of: (1) an expanding mass lesion (toruloma); (2) meningitis; or (3) meningoencephalitis. Headache, the main symptom in about 75% of affected patients, is usually either frontal, temporal or retroorbital. Other signs and symptoms include stiff neck, memory loss, agitation, psychoses, seizures, obtundation, cerebellar signs, hemiplegia, and increased intracranial pressure.[5]

Onset can be insidious; relapse is common. Cerebrospinal fluid may be clear, but the pressure is usually increased. When cells are present, lymphocytes predominate. With india ink, the yeast capsule appears as a transparent halo around spherical cells.

Although untreated cryptococcal infection has a high mortality, the course can sometimes be chronic. Beeson[6] described a case that lasted for 16 years. Treatment is with amphotericin or flucytosine (5-fluorocytosine).

At autopsy one may see: (1) exudate over the base of the brain and cerebellum with or without arteritis (meningitis); (2) extension along perivascular spaces with gray matter involvement with or without arteritis (meningioencephalitic form); or (3) granuloma, which may occur anywhere in the central nervous system.

Abnormal ocular findings are seen in about 40% of patients with cryptococcal meningitis. Okun and Butler[5] reviewed 36 cases and found 16 with ocular involvement. Blurred vision was the most common symptom. Other symptoms were retrobulbar pain, diplopia, and photophobia. Papilledema was the most common sign (12 of 16 patients). Other ocular signs were optic atrophy, sixth-nerve paresis, third-nerve paresis, nystagmus, homonymous field defect, and retinal exudates.

Kupfer and McDrane[7] found cryptococcal abscesses in optic nerves and tracts of three patients with papilledema and loss of visual acuity. They concluded that the visual loss was secondary to direct invasion by the organism of the nerve and tract.

In patients with papilledema who did have direct invasion of the visual pathways by the organism, there was no visual loss. This confirms the clinical finding that optic nerve decompression does not help to restore visual loss in cryptococcal meningitis.[8] When the organism invades the eye, granulomatous and necrotizing inflammatory changes are seen in the choroid, retina, and vitreous. Some patients can develop diffuse endophthalmitis.[9] Recently, one patient was described with an internuclear ophthalmoplegia and another with a supranuclear vertical gaze paresis.[10]

The finding of impaired pupillary and accommodative function in a patient with cryptococcal meningitis has not previously been reported to our knowledge. This finding is consistent with selective internal ophthalmoplegia secondary to involvement of the accommodative and pupillary fibers of both third nerves. These

TABLE

CLINICAL FINDINGS OF PATIENTS WITH SPONTANEOUS ORBITAL HEMORRHAGES

Patient No., Sex, Age, (yrs)	Eye	Onset	Initial Visual Acuity	Final Visual Acuity	Vomiting	Proptosis (mm)	External	Pain	Associated Factors	Pathology	Contrast Studies
1, M, 5	RE	24 hrs	6/12 (20/40)	6/12 (20/40)	Yes	10	Eyelid ecchymosis	Yes		Endothelial lined blood cyst	Venogram: mass lesion
2, F, 5	LE	Several days	6/60 (20/200)	6/6 (20/20)	Yes	5		No		Blood cyst	
3, F, 70	RE	24 hrs	6/9 (20/30)	6/9 (20/30)	No	4		No			Venogram: varix
4, F, 73	RE (recurred)	24 hrs	NLP	NLP	Yes	12	Eyelid ecchymosis	Yes			Venogram: bilateral varices
5, F, 2	RE	6 wks	Normal	Normal	No	5		No		Endothelial lined blood cyst	
6, F, 5	RE (recurred)	24 hrs	6/9 (20/30)	6/9 (20/30)	Yes	7	Eyelid ecchymosis	Yes			Venogram: varix
7, F, 47	LE (recurred)	24 hrs	6/5 (20/16)	6/5 (20/16)	No	2		No			Venogram: varix
8, F, 71	RE	24 hrs	6/9 (20/30)	6/6 (20/20)	No	5	Eyelid ecchymosis	Yes	Hypertension		Venogram: bilateral varices
9, F, 7	LE (recurred)	3 mos	6/9 (20/30)	6/9 (20/30)	No	16		Yes		Endothelial lined blood cyst	

10, M, 4	LE	4 days	HM	6/6 (20/20)	Yes	6		Yes		Blood cyst	Venogram: venous hypervascularity arteriogram-arterial hypervascularity
11, M, 6	LE (recurred)	24 hrs	6/6 (20/20)	6/6 (20/20)	Yes	10	Eyelid ecchymosis	Yes		Endothelial lined blood cyst	Venogram: mass lesion
12, F, 75	Both eyes	24 hrs	NLP	NLP	Yes	9	Eyelid ecchymosis	Yes	Anemia		
13, F, 80	RE	24 hrs	CF	CF	Yes	2		Yes			
14, F, 73	LE	24 hrs	6/9 (20/30)	6/9 (20/30)	Yes	3	Eyelid ecchymosis	Yes			
15, M, 35	RE (recurred)	24 hrs	6/9 (20/30)	6/9 (20/30)	No			Yes	von Willebrands disease Dental extraction		
16, F, 28	LE	24 hrs		6/6 (20/20)					Second stage of labor		Venogram: normal
17, M, 14	LE	24 hrs	6/6 (20/20)	6/6 (20/20)	No	1.5	Eyelid ecchymosis	No			Venogram: mass lesion

fibers are superficial in the oculomotor nerve situated just beneath the epineurium.[11]

Although peripheral oculomotor palsies usually have fascicular involvement, complete internal ophthalmoplegias have been reported in other basal meningitides such as tuberculosis, syphilis, and leprosy.[12]

Other causes for internal ophthalmoplegia could be lesions within the ciliary ganglion or brain stem. The absence of any other associated ocular signs, however, militates against lesions in these sites.

Finally, other causes of accommodation loss include diabetes, diptheria, botulism, viral disease, and cycloplegic drugs. None of these were found in our patient. The sixth nerve palsies could have been caused by either exudative meningitis or secondary to increased intracranial pressure.

Although cryptococcal meningitis is noted frequently in immunosuppressed patients with Hodgkin's disease, lymphoma, or leukemia, this infection is not seen frequently in systemic lupus erythematosus. In a review of 520 cases of systemic lupus erythematosus by Dubois and Tuffanelli,[13] no cases of cryptococcal infection were noted. In a review of 138 cases by Harvey,[14] only one case of cryptococcal meningitis was reported.

Approximately 25% of the patients with systemic lupus erythematosus in these series had nervous system involvement such as headache, irritability, psychosis, meningismus, and seizures. These cases, as well as our case, show the diagnostic difficulties encountered when a patient with systemic lupus erythematosus taking corticosteroids has abnormal neurologic findings. The possibilities that one must consider are: (1) primary central nervous system involvement from systemic lupus erythematosus[15]; (2) pseudotumor cerebri either from the systemic lupus erythematosus directly or secondary to corticosteroid treatment; and (3) opportunistic infection associated with chronic immunosuppression.

To our knowledge, this is only the tenth reported case of systemic lupus erythematosus associated with cryptococcal meningitis,[16-21] and is only the second case in which the diagnosis was made before death.[22]

SUMMARY

A 17-year old girl received prednisone and azathioprine for the treatment of systemic lupus erythematosus. She developed a fever and hallucinations 18 months later; cryptococcal meningitis was diagnosed. An internal ophthalmoplegia with loss of accommodation and dilation of the pupils developed together with bilateral lateral rectus palsy. Treatment with intravenous amphotericin resulted in disappearance of papilledema, muscle palsy, and internal ophthalmoplegia. We believe that the internal ophthalmoplegia was secondary to involvement of the accommodative and pupillary fibers of both third nerves at the base of the brain.

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LABORATORY ISOLATION TECHNIQUES IN HUMAN AND EXPERIMENTAL FUNGAL INFECTIONS

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The diagnosis of fungal infection of the eye has become more common in the past decade, partly because of improved laboratory techniques. The methods used to recover isolates have been derived from experience with systemic fungal infections, even though different organisms are likely to be recovered in ocular infections. Recent recommendations[1,2] regarding appropriate techniques for recovery of fungi were based on clinical experience in the southern United States, where *Fusarium solani* is the most frequent infecting organism.[1,2] In other geographic areas different fungi may predominate and it is important to determine whether these recommendations are broadly applicable.

We report herein our experience with a heterogenous group of human ocular isolates. Additionally, we have used an animal model of *F. solani* endophthalmitis to compare the efficacy of various fungal media.

MATERIAL AND METHODS

Clinical isolates—Specimens for fungal culture were obtained from the cornea of patients with suspected fungal infection by scraping or biopsy, or from the anterior chamber and vitreous by aspiration. This material was inoculated into four culture media, as follows: (1) trypticase soy agar with 5% defibrinated sheep blood; (2) Sabouraud's dextrose agar with 1% yeast extract freshly prepared without inhibitors, antibiotics, or buffers; (3) thioglycollate broth without indicator; and (4) brain-heart infusion broth medium (containing chloramphenicol, 25 μg/ml) without cyclohexamide; the broth (50 ml) was prepared in cotton-stoppered, 250-ml flasks to allow maximum aeration.

Corneal scrapings were inoculated on solid media by using a standard C-streak method.[3] Aqueous and vitreous samples were dropped on the surface of the solid media and their position marked by a wax pencil on the bottom surface of the dish. Growth occurring outside this defined area and off the C-streak was classified as contamination. Corneal material obtained with a sterile Kimura spatula was transferred to a sterile cotton-tipped applicator for immersion in liquid media. Both brain-heart infusion broth medium and thioglycollate broth were placed on a continuous rotary shaker after inoculation. All media were kept sealed and incubated at room temperature for four weeks before discarding. Slide cultures were done from all media showing growth on potato dextrose agar, cornmeal agar, Sabouraud's agar, and blood agar for further identification to genus, based on fruiting body morphology, and sensitivity testing. A Gram stain and Giemsa stain, and an occasional potassium hydroxide preparation were performed at the time of culture.

To investigate the possibility of contamination of liquid media (brain-heart infusion broth medium) during the inoculation process, the five following series of sham cultures were performed in the hospital eye clinic:

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Series 1—Ten unopened bottles of brain-heart infusion broth medium were inoculated for six weeks.

Series 2—Ten bottles were opened for 20 seconds then resealed and incubated for six weeks.

Series 3—Ten bottles were opened for 20 seconds; a sterile cotton-tipped applicator was inserted and the bottles were resealed and incubated for six weeks.

Series 4—A sterile Kimura spatula was wiped on a sterile cotton-tipped applicator which was then plunged into the opened broth bottle. Eighteen bottles treated in this manner were incubated for six weeks.

Series 5—Samples of tears from 13 normal patients were taken in a sterile capillary tube, which was then inserted into brain-heart infusion broth medium and incubated for six weeks.

Additionally, a second batch of ten series 3 sham cultures were performed in the ocular mycology laboratory.

Isolates from experimental fungal infection—An experimental model of *F. solani* endophthalmitis was used to compare the frequency of recovery in four culture media. This rat model has a recovery rate of greater than 90% two weeks after inoculation.[4]

Fusarium solani spores were injected into the anterior chamber of one eye of 55 rats by using a method previously described.[4] Two weeks later the animals were killed; the injected eye was enucleated under sterile conditions and ground to a fine suspension in a sterile tissue grinder. Aliquots of this suspension were then added to each of the four media to be tested (blood agar, Sabouraud's agar, thioglycollate broth, and brain-heart infusion broth medium containing 100 μg/ml of gentamicin). All cultures were incubated at room temperature for three weeks; the broth cultures were agitated on a rotary shaker. Media showing growth were subcultured to blood agar plates for organism identification by macroconidial morphology.[5] Twenty normal rat eyes were processed in a similar manner.

RESULTS

Twenty-six fungal isolates were recovered over a three-year period from 23 patients (there were three instances of double fungal infection). Apart from seven *Candida* species they were a heterogenous group (Table 1). Fifteen were isolated in brain-heart infusion broth medium alone, whereas three were isolated only on blood agar; two of the latter were *Candida* species. One isolate was recovered on Sabouraud's agar alone and only on five occasions was the Sabouraud's plate positive. One isolate was recovered in thioglycollate broth only. Overall, 21 of 26 fungal isolates (81%) were recovered in brain-heart infusion broth medium (Table 2).

The time required for the cultures to be positive varied from two to 19 days, with 25% of the isolates being recovered only after a period of 14 to 19 days (Table 3). Although *Candida* species and *F. solani* grew out rapidly, the other isolates usually did not.

The Giemsa or Gram stains were posi-

TABLE 1

HUMAN FUNGAL ISOLATES

Organism	No. of Isolates
Candida species	7
Fusarium solani	4
Aspergillus species	3
Cladosporium species	2
Torulopsis	1
Penicillium species	1
Paecilomyces species	1
Cephalosporium species	1
Alternaria	1
Epicoccum purpurescens	1
Helminthosporium	1
Ustilago	1
Stemphilium	1
Phoma	1

TABLE 2

FREQUENCY OF ISOLATE RECOVERY IN VARIOUS MEDIA

Isolates	Media: Brain-Heart	Blood Agar	Sabouraud's	Thioglycollate
20 in single culture medium	15	3	1	1
6 in multiple media	6	3	4	0
Total	21	6	5	1

tive for yeast forms or hyphal fragments in only six (25%) of the 23 cases. In five of these six, the isolates were recovered in only one of the four culture media used. In seven cases with negative smears, the isolates were recovered a second time on another medium, or during evisceration or keratoplasty. In three of these cases, 11 Giemsa stains and six potassium hydroxide preparations made at the time of culture were negative for fungal elements.

In the remaining ten cases, the organisms were recovered in only one medium and the diagnostic stains were negative. Nine of these appeared to be genuine fungal infections based on the clinical course and response to appropriate antifungal therapy. The remaining isolate (*Aspergillus*) was recognized as a contaminant on clinical grounds.

Only one of the sham cultures performed in the clinic became positive; *Penicillium* species was recovered from one of the series 4 bottles. The remainder of the sham cultures performed in the hospital eye clinic remained negative. Two of the ten sham cultures performed in the mycology laboratory grew *Penicillium* species.

Isolates from experimental fungal infection—Seventy-five rat eyes were submitted for culture. Of these, 55 had been inoculated with *F. solani* spores two weeks before enucleation. The remainder

TABLE 3

FREQUENCY OF ORGANISM ISOLATION, BY SPECIES AND MEDIA USED AND DAYS REQUIRED FOR RECOVERY

Total isolates	Organism	Media: Brain Heart	Blood Agar	Sabouraud's	Thioglycollate	Days to Positive Culture: Range	Mean
7	*Candida* species	5	5	4	0	2-4	2.9
4	*Fusarium solani*	4	0	0	0	2-4	3.5
3	*Aspergillus* species	2	0	0	1	2-14	7.7
2	*Cladosporium* species	2	0	0	0	3-14	8.5
10	Various species	8	1	1	0	3-19	10.4

were normal control eyes. None of the controls yielded positive bacterial or fungal cultures.

Of the 55 inoculated eyes, 51 (90%) yielded a positive culture of *F. solani* within two weeks; 43 (78%) of these were in brain-heart infusion broth medium, 40 (73%) were in thioglycollate broth, 37 (67%) on Sabouraud's agar. All eyes that yielded an isolate were culture-positive in brain-heart infusion broth, thioglycollate broth, or both. Eyes that were culture-negative in broth medium remained culture negative on Sabouraud's agar. Brain-heart infusion broth was the only positive culture medium in seven eyes and thioglycollate broth was the only positive culture medium in two other eyes. There was no bacterial or other fungal contamination in any culture medium in this study.

DISCUSSION

The etiologic agent in a suspected ocular infection can seldom be recognized clinically, and infections with more than one agent are by no means rare. Routine laboratory investigation should always include both bacterial and fungal media. Wilson and Sexton[6] discussed the advantages of a liquid medium for fungal isolation, and recommended the use of a shaker to ensure even nutrient distribution because of the prolonged period needed for growth to occur. Sabouraud's dextrose agar is preferred by some investigators[7,8]; others have recommended blood agar as a universal medium.[9,10]

In our laboratory the recovery rate for clinical isolates in brain-heart infusion broth medium has been impressive, but few isolates grew out on Sabouraud's dextrose agar or blood agar. In our experimental studies, brain-heart infusion broth medium was again superior to the solid media and thioglycollate broth was almost as good. The media inoculum in this experimental study contains mycelial fragments rather than the spores or germ tubes used in previous studies. This better approximates the clinical situation, since the mycelial phase is involved in invasive fungal disease.[11]

The incubation period necessary for fungal cultures is the subject of discussion.[11] In a large series, Forster found that the majority of isolates grew out in three to four days. *Fusarium solani* accounted for about 60% of his total isolates (personal communication, 1978), as compared to only 15% in our series. Although our experience with *Fusarium* species is identical to Forster's, one fourth of all our cultures became positive only after two to three weeks.

This prolonged period of incubation raises the possibility that contamination may have occurred, especially when only one culture medium becomes positive. We did identify one contaminant in this series of cases. The diagnosis in the remaining cases was consistent with fungal infection and the response to therapy was appropriate. Highly enriched media necessary for fungal isolation are especially liable to contamination during preparation and storage. Contamination may also occur during the culturing process or in the laboratory during the period of observation. Flasks and plates are sealed after inoculation to minimize subsequent contamination; however, the techniques used to sample the lesion have several steps, each of which involves the risk of a breach of sterility. In our sham culture study to examine this risk, we isolated a contaminant from the eye clinic study that apparently gained access to the medium during the 20 seconds that the flask was left open. The identification of two contaminants, presumably airborne, in the flasks inoculated in the mycology laboratory further emphasizes this problem. C-streak inoculation of solid media has been advocated as a means of identifying such spurious isolates. However, contamination by fungi present in the tear film may also occur. Although they do not contribute to the disease process under investigation, these commensal organisms could easily become incorporated in the inoculum. In the small number of patients sampled we did not isolate fungi from the tear film, but other investigators[12,13] have found that from 3% to 30% of individuals carry fungi in the conjunctival sac in the absence of disease.

A positive smear obtained from the lesion at the time of culture is important confirming evidence that the isolate is not a contaminant. However, correlation between smear and culture data is not exact and we have noted a tendency by inexperienced observers to read these smears incorrectly as positive smears. We demonstrated the presence of fungal elements on smear in only 26% of our patients with positive cultures. In two previous reports[9,14] about half the patients with culture-proven fungal infections had positive smears. In the only other report,[1] this correlation was not specifically examined. However, the percentage of positive smears appeared to be higher.

The recovery of an isolate on a second medium places the diagnosis on a firmer basis, but in our experience this can be expected in only a small percentage of cases. Most fungi tend to be invasive, with the major portion of mycelial growth occurring within tissue rather than at the surface of the lesion.[11,15] With the exception of superficial and overwhelming infections, only a small amount of viable fungal material may be accessible to scraping techniques. Thus, the probability of inoculating more than one medium and also demonstrating fungal elements on smear may be low. The amount of fungal material obtained by scraping and the relative difficulty in obtaining a positive culture cannot be used as an indication of the extent or seriousness of fungal invasion.

Despite our favorable results with brain-heart infusion broth medium, we believe that reliance on this or any single medium is unwise. The use of several media increases the chances of isolate recovery.[1,10] When a fungal infection is suspected we inoculate both liquid and solid media prepared in the manner previously described. All media are incubated at room temperature and are not discarded for at least four weeks. If one medium becomes positive the remaining cultures are observed for confirmation of the original isolate, and also to ensure that another agent is not present. Our studies indicate that investigators must always be alert to the possibility of contamination.

SUMMARY

In laboratory experience with a heterogenous group of 26 human ocular fungal isolates, brain-heart infusion broth proved to be the most useful medium for isolation. Although *Candida* and *Fusarium* species grew out within four days of inoculation, one fourth of the cultures did not become positive until 14 to 19 days had elapsed.

In an animal model of endophthalmitis due to *F. solani* infection, brain-heart infusion broth again was the most useful.

The highly nutritious media used for fungal isolation are prone to contamination by organisms that are difficult to distinguish from true pathogens. Sham culture studies demonstrated that this contamination can easily occur during the process of sampling the lesion and inoculating the media.

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POLYPOID SINUSITIS MIMICKING ORBITAL MALIGNANCY

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The rapid onset of unilateral proptosis with marked exophthalmos, eye deviation, and orbital bony erosion are all signs of orbital malignancy. We describe herein a patient with a history of nasal inflammatory polyps who had a deviating, proptosed, exophthalmic right eye, with bony erosion. Initial diagnosis was orbital malignancy, which was found to be polypoid sinusitis.

CASE REPORT

A 15-year-old boy was referred here for evaluation of proptosis of the right eye, noted two months before admission. Because of an eight-year history of recurrent sinusitis, the patient had had bilateral Caldwell-Luc procedures, with decompression of the maxillary sinus via nasal antral windows one year before admission. On examination the patient had right proptosis with exophthalmos of 7 mm, as compared to the left eye by Hertel measurements, with the globe displaced inferotemporally (Fig. 1). The onset was painless, and the patient denied diplopia or visual disturbances. The proptosis was nontender and noncompressible. Extraocular movements and levator muscle function were normal in both eyes. Palpation revealed a 2 × 3-cm firm mass, located superonasally in the right orbit, apparently fixed to deeper tissues. Results of the rest of the ocular examination were unremarkable. Nasal examination revealed a soft, flesh-colored, crusted mass arising from the posterolateral right nasal cavity with deviation of the nasal septum to the left side.

Complete blood cell count, blood chemistries, urinalysis, and chest x-rays were within normal limits. The patient was afebrile. Radiologic examination, including orbital tomograms, showed opacification of both maxillary antra, ethmoidal and frontal sinuses with a right nasal mass, and erosion of the medial wall of the right orbit. The tomograms confirmed these findings and suggested erosion of the right orbital floor posteriorly. Computed axial tomography (Fig. 2) revealed diffuse opacification of the right ethmoid and right nasal area extending posteriorly, both above and below the right proptotic globe. There was no enhancement of the tumor with contrast added to the computed axial tomography. Ultrasound examination (Fig. 3) revealed areas of sound attenuation and reflectance in the mass. A biopsy of the nose suggested nasal polyps. With the erosion and marked proptosis as well as rapid onset of this lesion, a nasal antral decompression with a right percutaneous ethmoidectomy and anteronasal orbitotomy were performed. The mass was excised completely and comprised a firm, tan hemorrhagic tissue, including a 3 × 3 × 2-cm multilobulated mass from beneath the periosteum of the right anteronasal orbit that emanated through a 1-cm diameter defect in the lamina papyracea. The right orbital dissec-

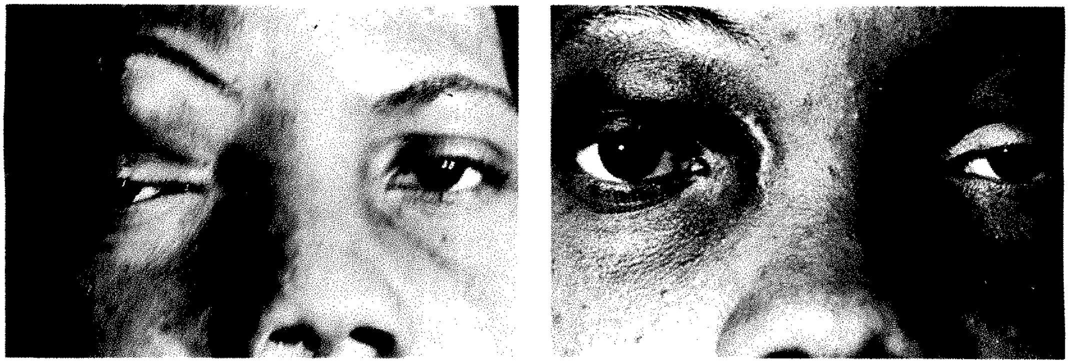

Fig. 1 (Rawlings, Olson, and Kaufman). Preoperative (left) and postoperative (right) appearance of patient with polypoid sinusitis simulating orbital malignancy.

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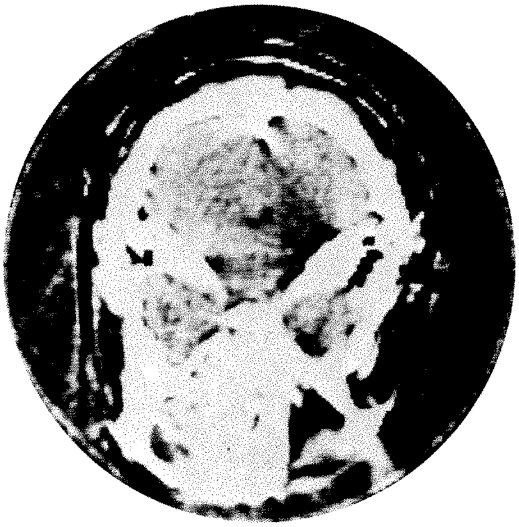

Fig. 2 (Rawlings, Olson, and Kaufman). Computed axial tomography scan showing a right nasal antral mass extending into the right medial orbit with the globe displaced anterolaterally.

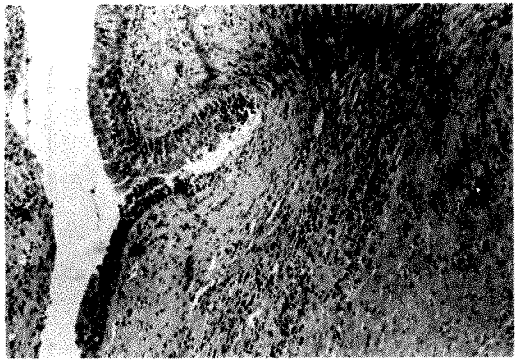

Fig. 4 (Rawlings, Olson, and Kaufman). Tissue from right anterior orbitotomy showing hyperplastic pseudostratified columnar epithelium with marked edema and acute and chronic inflammatory cells (hematoxylin and eosin, ×75).

tion was done deep to the periosteum. There were no other defects in the orbital wall. The postoperative course was uneventful, and the patient has done well (Fig. 1). The pathologic specimen showed "numerous polypoid structures covered by focally hyperplastic pseudostratified ciliated columnar epithelium. There was an increase in mucus producing cells and mucus filled, dilated glands in the stroma. The stroma is edematous and shows numerous acute and chronic inflammatory cells with a predominance of lymphocytes and plasma cells. Very few eosinophils are present. Neovascularization, fibrosis, and bone spiculi are also present. These microscopic findings are those of nasal polyps" (Figs. 4 and 5).

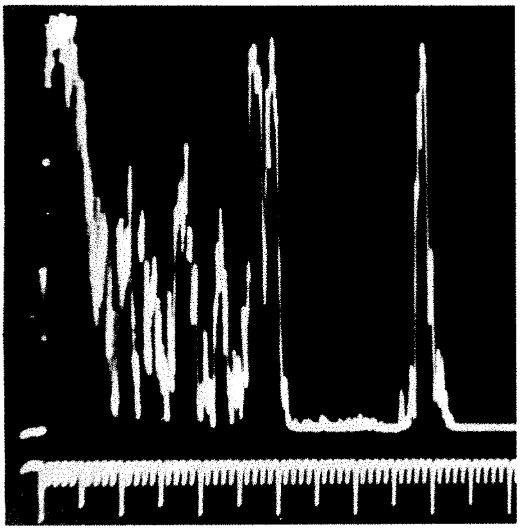

Fig. 3 (Rawlings, Olson, and Kaufman). A-scan ultrasonogram directly over the orbital mass showing moderately high internal reflectivity of the tumor.

DISCUSSION

Whereas a mucocele is a relatively common cause of progressive unilateral exophthalmos, this is, to our knowledge, the first reported case of rapidly progressive

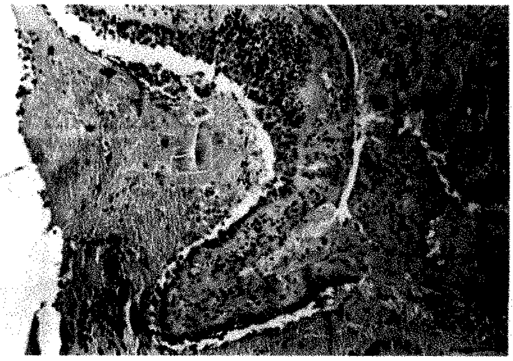

Fig. 5 (Rawlings, Olson, and Kaufman). Sections of tissue from right ethmoidectomy showing bone spiculi interspersed with dilated mucus glands and fibrosis in the polyp stroma (hematoxylin and eosin, ×75).

exophthalmos caused entirely by polypoid sinusitis. Sinus disease with mucocele has been reported to cause approximately 10% of all cases of proptosis via extension from the frontal sinus and has been reported as being spongy when palpated.[1,2] In this case, the clinical picture of bony erosion of the orbit and rapidly progressing proptosis with apparent fixation of the mass to the bony orbit was indicative of a malignant process. The mass had a relatively firm consistency by palpation and, in combination with an ipsilateral nasal mass, indicated a possibly fatal prognosis. The pathologic specimens, however, are unequivocal and consistent throughout, showing this lesion to be sinus polyposis. Cystic structures were not identified at surgery or on the gross pathology report, on computed axial tomography, or on tomography. The definitive evidence was a low-power view of the specimen (Fig. 6) showing that the tissue (Fig. 5) was the substantia propria of polyps and not densely fibrous tissue from the wall of a mucocele.

The age of the patient should be considered when diagnosing unilateral proptosis. In the adult, dysthyroid ophthalmopathy and malignancy are the major causes of unilateral exophthalmos, followed by extension of local disease and trauma.[3-7] Most of the patients included in these surveys are adults; however, there is general agreement that, in adults, about 65% of unilateral proptosis is the result of a primary orbital problem, with 25% from extension of a local disease process, and 10% from metastatic disease.[8]

The most common cause of rapidly progressing unilateral exophthalmos is rhabdomyosarcoma, and a biopsy specimen should be taken immediately and the lesion treated with radiation therapy to avoid the consequence of actual expulsion of the globe.[9,10] This diagnosis would, however, be unusual in our 15-year-old patient. Because of the simultaneous orbital and nasal masses, inverted nasal papilloma, nasopharyngeal angioma, and juvenile nasopharyngeal carcinoma were also considered in the differential diagnosis.

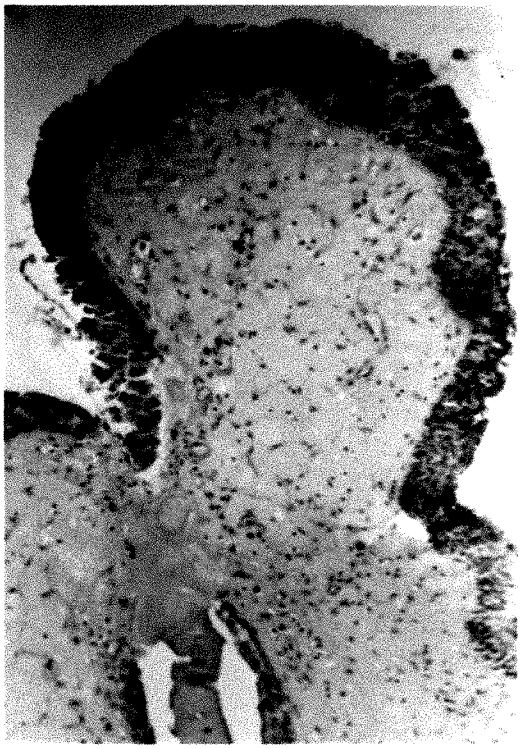

Fig. 6 (Rawlings, Olson, and Kaufman). Photomicrograph of specimen from right anterior orbitotomy showing substantia propria of polyps invading orbit (hematoxylin and eosin, ×130).

Inverted nasal papilloma[11] usually occurs spontaneously in older patients, 70% occurring in men over 40 years old. These are true neoplastic lesions, usually unilateral and of firm consistency. The bony destruction that can occur with these tumors is less common than bone attenuation, but the disease can mimic malignancy. Many consider inverted nasal papilloma to be a low-grade malignancy. Our patient's age, orbital extension, and history of inflammatory sinus disease were not consistent with this diagnosis.

Nasopharyngeal angioma (juvenile angiofibroma) has a 90% rate of occurrence in males with an average age of 15 years.[12] This tumor frequently produces a superior orbital fissure syndrome and mimics malignancy by intracranial spread and bony erosion. These tumors characteristically bleed profusely when a biopsy specimen is taken and have marked enhancement during computed axial tomography with contrast. The absence of contrast enhancement during computed axial tomography helped rule out this possibility preoperatively in our patient.

Nasopharyngeal carcinoma [13] also has a higher male incidence (75%), with most cases occurring in patients over age 40 years. Juvenile nasopharyngeal carcinoma is not rare, and a small age peak seems to occur at about age 15 years. This tumor usually originates from the lateral nasal wall and frequently produces a cavernous sinus syndrome as the orbital apex is eroded. Our case mimicked nasopharyngeal carcinoma, although the history and bilaterality of sinus disease were not typical of malignancy.

Our case shows that polypoid sinusitis can mimic rapidly progressing exophthalmos and proptosis, particularly in a patient already known to have severe sinus disease. In our case, the surgical treatment was greatly simplified because orbital extension was entirely subperiosteal.

SUMMARY

A 15-year-old boy developed a rapidly progressive exophthalmos of 6 mm within two months. Radiologic evidence showed bony erosion, a palpable firm mass in the right upper eyelid, and mass blocking the right nares. This tumor was found to be polypoid sinusitis emanating through the lamina papyracea.

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ACUTE OCULAR LEUKEMIA

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Leukemias and lymphomas involve the eye in various ways. Allen and Straatsma[1] reviewed the ocular sites and frequency of involvement and found that the choroid, especially in the posteior pole, is the most frequent site of infiltration. Typically, the choroid shows diffuse thickening. When the retina is affected it is by localized perivascular infiltrations, microinfarction, superficial and deep hemorrhage, and discrete tumor infiltrations.

Burns, Blodi, and Williamson[2] described a case of bilateral central serous retinopathy in a patient with acute lymphoblastic leukemia. The decreased vision and serous detachments occurred during a relapse of leukemia. Zimmerman and Thoreson[3] described a similar case with unilateral serous detachment.

Clayman and associates[4] described two children with acute leukemia who developed visual loss and a peculiar clumping of the retinal pigment epithelium. In both children, this change was preceded by generalized relapse and papilledema. Associated with the localized hyperplasia were areas of retinal pigment epithelium cell destruction.

We describe herein a patient with an unusual leukemia in which visual impairment was the symptom. The disease was not diagnosed until after death.

CASE REPORT

A 71-year-old woman was first seen on Jan. 10, 1977, complaining of visual loss of four days' duration. No pain or diplopia were present. Visual acuity was hand motions in the right eye and 6/30 (20/100) in the left eye. Refraction showed hyperopia of 7 diopters; her spectacle lens power was 6 diopters. Ophthalmoscopic examination of the right eye showed a normal optic nerve with slightly narrowed retinal vessels. The retina was moderately raised in the macular area, and the detachment extended inferiorly to the periphery. There were no retinal holes. The ora serrata was easily seen inferiorly and temporally without scleral depression, suggesting a ciliochoroidal effusion. The subretinal fluid shifted readily with posture changes. The left eye showed a shallow detachment of the posterior pole. The inferotemporal periphery showed no retinal elevation but a distinct choroidal detachment.

Medical history included a 50-lb weight loss over the previous two years, which was attributed by the patient to anxiety. The family had noted increasing mental depression. Family history was noncontributory except for systemic histoplasmosis in a daughter.

Physical examination disclosed a low-grade bruit over the left carotid artery. Otherwise, the cardiovascular system was normal. Results of neurologic examination were negative.

Fluorescein angiography of both eyes showed a normal circulation pattern in the retinal vessels with no evidence of leakage. Stereophotographs confirmed the retinal elevation. Beneath the retina in the early phases of the angiogram, myriad hyperfluorescent pinpoint dots were seen (Fig. 1). With time, these dots became more diffuse, leaking dye into the subretinal space (Fig. 2). The peripheral choroidal detachment did not glow.

The patient was admitted for an extensive medical examination. An erythrocyte sedimentation rate was 57 mm/hr (Wintrobe), but a temporal artery biopsy was negative. A brain scan and skull series indicated no intracranial lesions. With no specific cause for the bilateral retinal detachments evident, the patient was given a trial of systemic corticosteroids, and was discharged.

She was readmitted to the psychiatric service on Feb. 23, 1977, because of deteriorating mental status and increasing depression. This progressed to disorientation and aphasia, and she was transferred to the medical service.

Despite an intensive medical examination, no cause for the neurologic changes was found and her status continued to deteriorate. Repeat sedimentation rate was 59 mm/hr and a repeat temporal artery biopsy was negative. She began to have spiking fevers that continued despite antibiotics for a urinary tract infection. An electroencephalogram showed diffuse changes suggestive of encephalopathy. A lumbar puncture showed an increased protein

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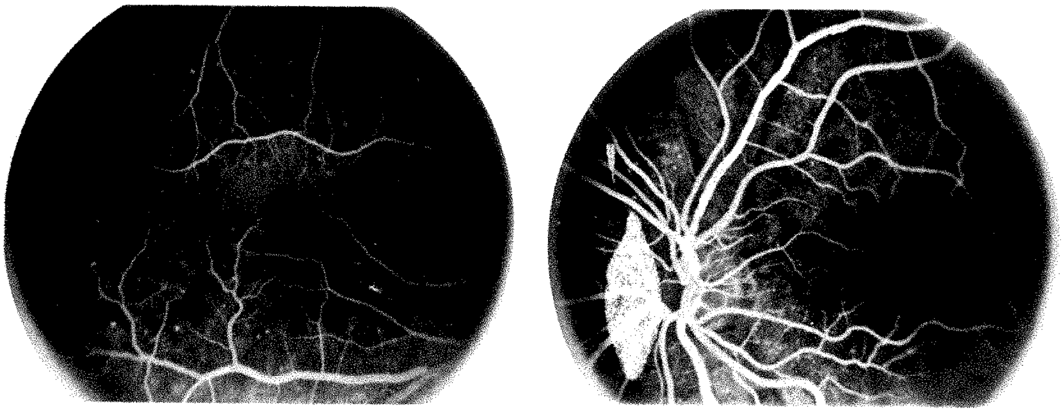

Fig. 1 (Kincaid, Green, and Kelley). Fluorescein transit in early venous phase (28 seconds) of right and left eyes showing a myriad of pinpoint areas of leakage at level of retinal pigment epithelium.

of 180 mg/ml but no pleocytosis. Opening pressure was 314 mm Hg. Anemia was evident, with a hemoglobin of 7.3 g/100 ml and hematocrit of 22.3%. The white blood cell count was increased at 19,800 mm^3 with no abnormal cells. Bone marrow biopsy revealed immature cells and some unidentified cells that were believed at the time to be a possible metastatic tumor. Despite further tests, no primary tumor was found. She continued to deteriorate, and one day before death developed myoclonic seizures. She died April 19, 1977.

At autopsy, extensive tumor infiltration of the pituitary gland, brain, liver, kidney, and adrenals was observed. The bone marrow, however, showed patchy, rather than diffuse, involvement. The cellular distribution did not precisely fit that of either a lymphoma or a leukemia. A modified esterase test[5] indicated possible myeloid origin of the cells.

In both eyes the conjunctiva, cornea, anterior chamber, iris, lens, and trabecular meshwork were all normal. The choroid was moderately distended by a diffuse infiltrate of neoplastic cells, most of which were located within choroidal vessels (Fig. 3). These cells were pleomorphic, with scant eosinophilic cytoplasm and large clear nuclei with one or more prominent dark nucleoli. The cells were loosely packed and had up to five mitoses per 40 high-power fields. A small, flat serous detachment of the retina was present in the macular area (Fig. 4). Additionally, diffuse focal retinal pigment epithelium changes were evident. The retinal pigment epithelium was intact throughout but showed areas of relative depigmentation (Fig. 5), and proliferation in both a nodular (Fig. 6) and a double-row configuration. A few areas of small retinal pigment epithelium serous detachment were seen (Fig. 7).

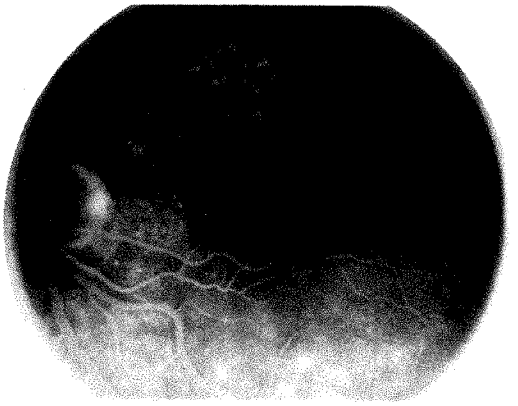

Fig. 2 (Kincaid, Green, and Kelley). Later frame in the fluorescein study of left eye (98 seconds) shows more diffuse staining and leakage of dye into the subretinal space.

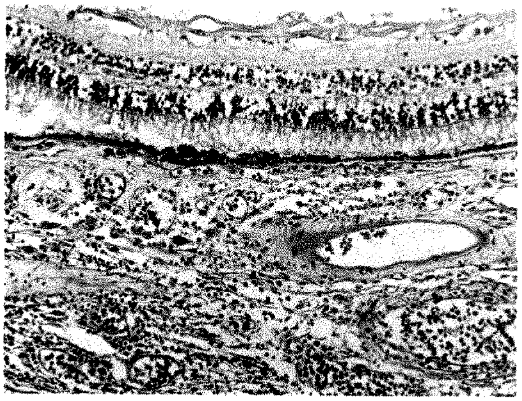

Fig. 3 (Kincaid, Green, and Kelley). Thickening of choroid by infiltration of tumor cells, mainly located within the vascular spaces (hematoxylin and eosin, ×160).

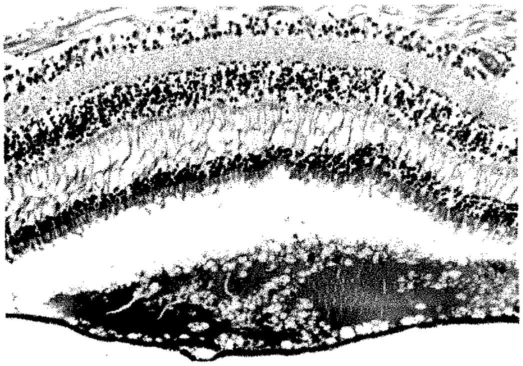

Fig. 4 (Kincaid, Green, and Kelley). Serous detachment of retina in macular area (hematoxylin and eosin, ×140).

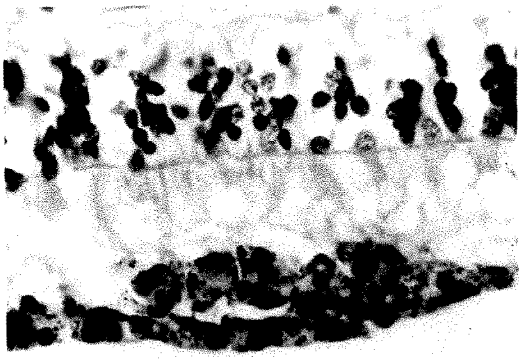

Fig. 6 (Kincaid, Green, and Kelley). Small area of hyperplasia of the retinal pigment epithelium (hematoxylin and eosin, ×850).

Discussion

When first examined, we thought the patient's symptoms were consistent with Vogt-Koyanagi-Harada syndrome.[6-8] The ophthalmoscopic findings, angiograms, and mental changes supported this diagnosis. Her age, fair pigmentary characteristics, and the absence of other systemic signs such as dysacousia, vitiligo, poliosis, and meningeal irritation were not consistent with this diagnosis. She showed no evidence of uveitis in the anterior chamber or vitreous.

The diagnosis of ciliochoroidal effusion (uveal effusion) was also considered. Consistent with this diagnosis were the absence of uveitis, the serous retinal detachments, ciliochoroidal effusion, and the increased spinal fluid pressure and protein.[9-11] However, this is a disease found almost exclusively in middle-aged males. Fluorescein angiograms in uveal effusion are consistent with stasis of the choroidal vasculature and show larger and fine pigment mottling.[12]

The fluorescein findings found in this patient were consistent with a diffuse choroidal infiltration. The angiogram

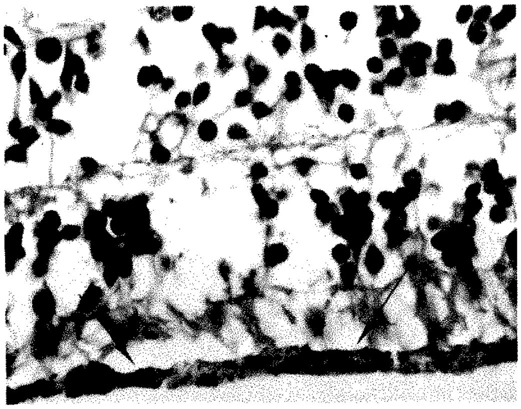

Fig. 5 (Kincaid, Green, and Kelley). Area of retinal pigment epithelium with reduced number of melanin granules (between arrows) (hematoxylin and eosin, ×875).

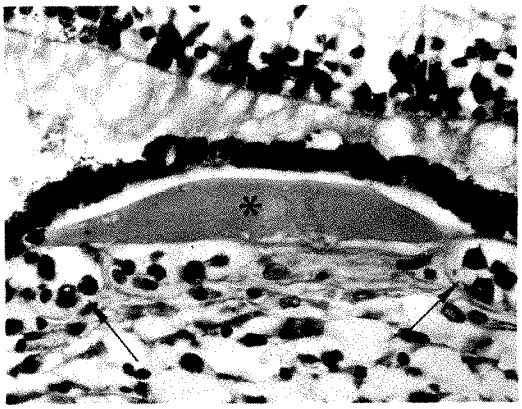

Fig. 7 (Kincaid, Green, and Kelley). Small serous detachment (asterisk) of retinal pigment epithelium. Tumor cells (arrows) are present in the choriocapillaris (hematoxylin and eosin, ×650).

showed myriad points at the level of the retinal pigment epithelium. Those fluorescein changes, which we call "Milky-Way pattern," are seen in malignant melanomas of the choroid,[13] metastatic tumors to the choroid,[14] posterior scleritis,[15] and Vogt-Koyanagi-Harada syndrome.[8] Single or multiple retinal pigment epithelium leakage points may also be seen in idiopathic central serous choroidopathy.[16] With the exception of the central serous choroidopathy, in which the pathology is uncertain, these conditions are all characterized by choroidal infiltration, either diffuse or nodular.

Gass[17] described a patient with myelomonocytic leukemia who had a discrete choroidal mass in the left eye. This 59-year-old woman had a serous detachment and complained of blurred vision and metamorphopsia. The fluorescein angiogram showed pinpoint hyperfluorescence in the area of the choroidal mass. The increase was confirmed by stereophotographs. In our patient the diagnosis was obscured because the choroid was diffusely infiltrated and there were no discrete elevations. The problem is similar to that of diagnosing a flat melanoma[18] and diffuse intraocular inflammatory pseudotumor.[19] In the case described by Gass there were several placoid areas of staining at the level of the retinal pigment epithelium, which Gass believed were areas of pigment epithelial detachment. This was not evident in our case, but pathologic examination disclosed small areas of retinal pigment epithelium detachment.

We believe that the retinal pigment epithelium changes and serous retinal detachments in our case were caused by leukemic infiltration in the choroid with primary or compressive involvement of the choriocapillaris. The infiltrate interferes with the blood supply to the retinal pigment epithelium and leads to small localized areas of disruption. Another unusual feature of our case, as in the one described by Gass,[19] is that the ocular involvement was the initial symptom of the disease. In most other reported cases, ocular involvement has appeared as part of an acute exacerbation later in the disease course.

SUMMARY

A 71-year-old woman had a history of recent weight loss and bilateral decreased visual acuity, bilateral serous detachment, and mental depression. Fluorescein angiograms showed a myriad of retinal pigment epithelial leakage points.

Despite extensive evaluation, the cause of her weight loss and ocular process remained uncertain until her death, when postmortem examination revealed leukemic infiltrates of many organs, including the choroid.

After death, we correlated the clinical signs and fluorescein angiograms with the histopathologic findings. This case shows that choroidal disease may be a symptom of undetected leukemia.

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OPHTHALMIC MINIATURE

. . . This King Cymbeline, who has now made himself overlord of the greater part of the island, gives a generous welcome to Roman-French traders and even to Greek doctors, especially oculists, for the British seem to suffer pretty severely from ophthalmia, owing to the marshiness of the country.

Robert Graves, *Claudius the God*
New York, Vintage Books, 1935

PERIPHERAL RHEUMATOID ULCERATION AND EVIDENCE FOR CONJUNCTIVAL COLLAGENASE PRODUCTION

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Patients with rheumatoid arthritis may have peripheral corneal ulcerations.[1,2] The ulcers are usually located inferiorly and are not accompanied by hyperemia or vascularization. The ulcers may remain indolent or progress to extreme thinning and ultimately to perforation.

Recently, excision of limbal conjunctiva has been advocated in treatment of progressive peripheral corneal ulcers.[3,4] This technique has been successful in treatment of Mooren's ulcer and peripheral ulceration associated with rheumatoid arthritis. The limbal conjunctiva may be examined for a variety of properties that include cellular morphology, immunoglobin deposition, and production of degradative enzymes. We describe herein the successful treatment of two rheumatoid patients with marked progressive corneal thinning by conjunctival resection, and provide evidence for collagenase production in the limbal conjunctiva proximal to the ulcerations.

CASE REPORTS

Case 1—A 71-year-old man with a long history of rheumatoid arthritis had severe rheumatoid changes of both upper extremities with extreme ulnar deviation of the fingers and rheumatoid nodules of the elbows. His arthritic involvement of the lower extremities was so severe that he had a total left knee replacement before the present treatment.

Ocular examination revealed visual acuity of R.E.: 6/120 (20/400) and L.E.: 6/12 (20/40). The right eye had a large crescentric peripheral corneal ulcer at the inferonasal corneoscleral limbus (Fig. 1). The ulcer extended approximately 90% through the corneal stroma; there was no evidence of corneal vascularization. Although the ulcer pooled fluorescein, there was no rose bengal or fluorescein staining of the cornea. The anterior chamber was deep and clear although there was evidence of old keratic precipitates on the posterior cornea. There were also two posterior synechiae to the lens. There was 2+ nuclear sclerosis and 3+ posterior subcapsular cataract in the right eye. Applanation pressure was 18 mm Hg bilaterally. Schirmer's testing revealed 8 mm of wetting in both eyes. The remainder of the ocular examination was normal. The patient had a positive rheumatoid factor and positive antinuclear antibodies with a titer of 1:160.

Because of the extreme corneal thinning and possible perforation, the patient was operated on and a 5.0-mm conjunctival resection of the adjacent conjunctiva was performed without complications. Postoperatively the ulcer filled in with a fibrous coagulum and healed within two to three weeks (Fig. 2).

Three conjunctival specimens were assayed for collagenase activity. One sample was taken at the corneosceral limbus proximal to the site of ulceration. The second (nonlimbal) specimen was taken 7 mm distal to the first sample. The third tissue sample assayed was normal (control) conjunctiva taken from a site 180 degrees opposite to the ulcer.

Case 2—A 68-year old man with a long history of rheumatoid arthritis had severe crippling disease of the fingers with ulnar deviation and marked deformities of the fingers. Visual acuity was R.E.: 6/60 (20/200) and L.E.: 6/18 (20/60). The eyes were

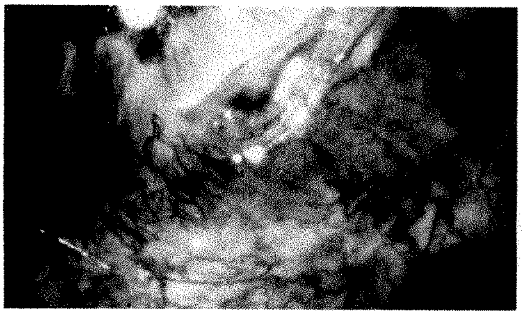

Fig. 1 (Eiferman, Carothers, and Yankeelov). Case 1. Cresentric peripheral corneal ulcer at the inferonasal corneoscleral limbus. The ulcer extends approximately 90% through the corneal stroma.

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This study was supported by USPHS Grant No. EY 00969.

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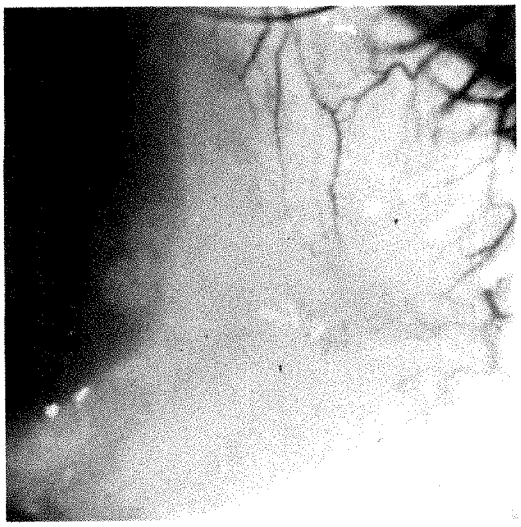

Fig. 2 (Eiferman, Carothers, and Yankeelov). Case 1. After conjunctival resection. Postoperatively the ulcer filled in and healed within two to three weeks.

white and quiet. An inferior marginal ulcer extended from approximately 6:30 to 8:30 o'clock on the right eye (Fig. 3). The ulcer was approximately 90% through the corneal substance. There was no evidence of abnormal limbal or corneal vascularization. The anterior chamber was deep and clear and the applanation pressure was 16 mm Hg in both eyes. There was 3+ nuclear sclerosis in the right eye and 1+ nuclear sclerosis in the left eye. Schirmer testing produced 8 mm of wetting in both eyes. Although the ulcer stained, there was no additional rose bengal or fluorescein staining. His serum was positive for both rheumatoid factor and antinuclear antibodies.

Because of the corneal thinning, the patient was operated on and a 5.0-mm conjunctival resection of the adjacent conjunctiva was performed. Three conjunctival specimens were removed and assayed for collagenase activity as in Case 1. Postoperatively the patient did well and the ulcer filled in over the next few weeks (Fig. 4).

MATERIAL AND METHODS

Lathyritic rat skin collagen was prepared as described by Yankeelov, Wacker, and Schweri.[5] Assay dishes were prepared by suspending 1.2 g of Sea Plaque agarose in 60 ml of Tris buffer (pH 7.6, 20°C containing 0.05 M Tris, 0.4 M NaCl, 3 mM $CaCl_2$) and 0.04% sodium azide. The mixture was degassed for 15 minutes in a suction flask at 20°C and then heated to above 75°C to dissolve the agarose. The hot solution (48 ml) was transferred to a 100-ml capacity water-jacketed beaker at 37°C. The solution was stirred constantly with a Z-shaped stirring blade. After the agarose had equilibrated to 37°C, an

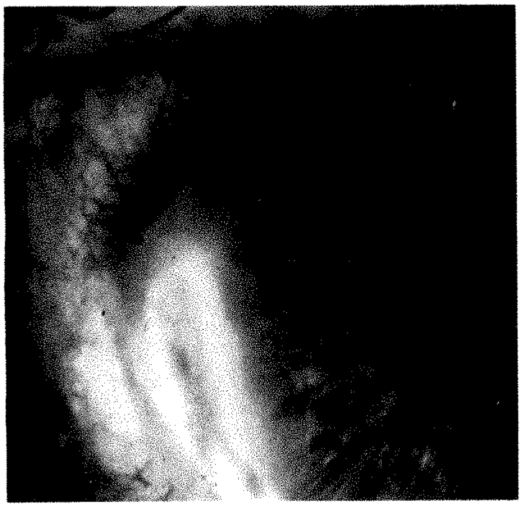

Fig. 3 (Eiferman, Carothers, and Yankeelov). Case 2. Inferior marginal ulcer from 6:30 to 8:30 o'clock of right eye. The ulcer extends through approximately 90% of the corneal substance.

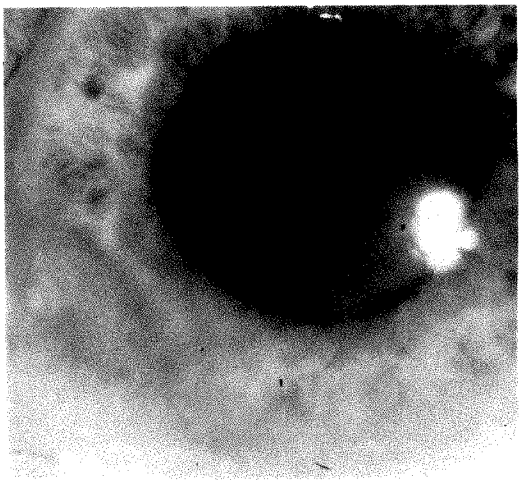

Fig. 4 (Eiferman, Carothers, and Yankeelov). Case 2. After conjunctival resection. Postoperatively the ulcer filled in and the patient did well.

equal volume of 0.2% collagen (20°C) was added by drops to the agarose over a period of seven minutes. Stirring was maintained at a rate that did not introduce bubbles. The mixture was allowed to stir for 30 minutes during which time finely divided collagen fibrils were produced. Aliquots (6.0 ml) of the mixture were transferred rapidly to 50 × 12 mm petri dishes. Each dish was allowed to stand open for two hours. The set plates were covered and refrigerated.

Rectangular pieces of resected conjunctiva 2 to 3 mm on an edge, previously maintained for less than six hours at 20°C in balanced salt solution (BSS), were placed in nominal 3.0-mm wells that had been punched in the assay dish matrix. Air spaces were eliminated by addition of balanced salt solution. A well containing only balanced salt solution was included as a control. The dishes were covered and incubated near 100% humidity at 32°C for a period of 96 hours. Photography was done according to the method of Hunter.[6]

As reported previously[5] the use of lathyritic collagen supported in agarose matrix eliminates the mechanical instability inherent in unsupported collagen gels. The low setting temperature of Sea Plaque agarose (<30°C) permits formation of the gel without the threat of substrate denaturation. The reversible, temperature-dependent fibril formation characteristic of lathyritic collagen may be used to advantage by manipulation of assay conditions to allow digestion of either collagen monomers or collagen fibrils, although the boundaries are sharpest after digestion of collagen monomers. Fibril formation may be induced by a brief incubation at physiologic temperatures. Lathyritic collagen fibrils are completely resolubilized by refrigeration, and the assay plates may be safely stored in this manner for several weeks without damage to the sensitivity of the assay.

Previous studies[5] have shown the assay system to be valuable in testing inhibitors of tadpole collagenase. The specificity of the assay for collagenase was confirmed because trypsin samples of 0.2 mg/ml produced no digestion. Trypsin at high concentrations (3 to 6 mg/ml) produced significant zones of lysis that were much more diffuse than those produced by tissue collagenase.

Biopsy specimens of the conjunctiva adjacent to the ulcer as well as 180 degrees opposite to the ulcer were taken. The tissue was frozen on a cryostat and cut into 4-μ sections. Tissue was washed in phosphate buffered saline (pH 7.2) and then directly stained using fluorescein conjugated antiserum to human IgA, IgM, IgG, IgE, and C′3. The specimens were incubated for 30 minutes at room temperature and rinsed with phosphate-buffered saline. The sections were mounted with buffered saline and examined under a fluorescence microscope and photographs were taken. Indirect immunofluorescence was performed by using normal human cornea and conjunctiva obtained within one hour of death as a normal control. These tissues were frozen and cut into 4-μ sections on the cryostat. They were air-dried and overlaid with twofold dilutions of the patient's sera for 30 minutes. The tissue was then washed with phosphate-buffered saline and incubated with fluorescein labeled antiserum to IgG, IgE, IgA, IgM, and complement. The tissue was then washed and mounted in a fluorescence microscope. Specimens were also embedded in paraffin and processed for routine light microscopy.

RESULTS

Light microscopy revealed the epithelium of the conjunctiva to be intact. There was a minimal inflammatory response in the substantia propria of both proximal and distal conjunctiva that consisted of round cells and polymorphonuclear leukocytes. There was no increase in plasma cells seen. Similarly there was no evidence of vasculitis. The control specimen was unremarkable.

Direct immunofluorescence revealed a bright band of IgG along the basement membrane of the epithelium. IgA, IgM, IgD, IgE, and complement were all negative. Indirect immunofluorescence was negative, revealing no evidence of circulating corneal or conjunctival antibodies.

The collagenase assay results are shown in Figures 5 to 8. Figure 5 shows the results of the collagenase assay performed on normal conjunctiva taken from routine cataract patients. There was no evidence for collagenase production by normal tissue.

Figure 6 shows the relative positions of the conjunctival samples assayed for collagenase from both patients; the limbal conjunctiva adjacent to the ulcer (A), the conjunctiva 7 mm from the corneoscleral limbus (B), and the site of normal conjunctiva 180 degrees removed from the location of the ulcer (C).

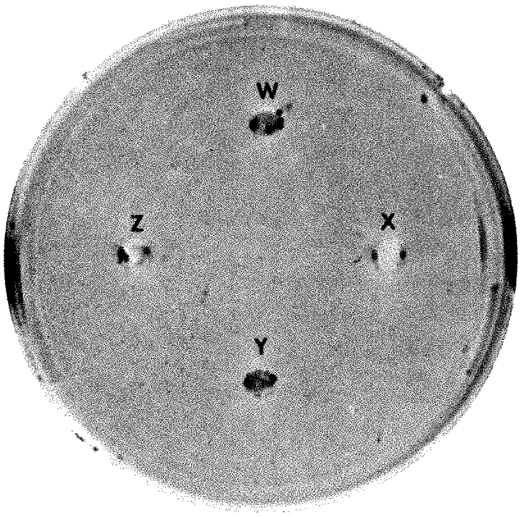

Fig. 5 (Eiferman, Carothers, and Yankeelov). Radial diffusion assay for collagenase performed on normal conjunctiva taken from cataract patients. No collagenolytic activity is seen. Wells marked X and Z contained balanced salt solution. Wells marked W and Y contain conjunctiva.

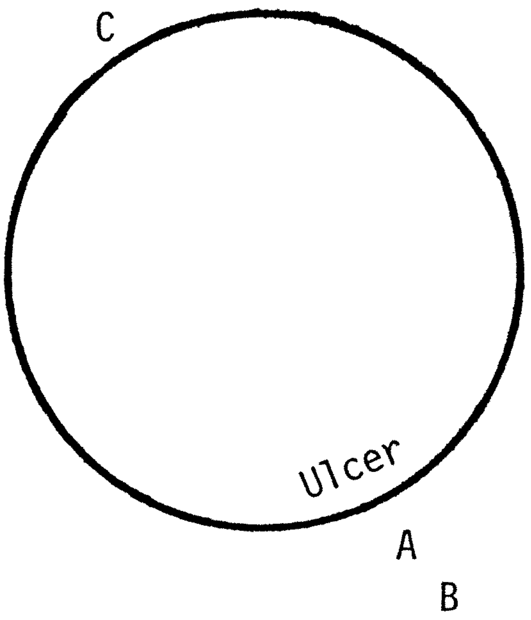

Fig. 6 (Eiferman, Carothers, and Yankeelov). Schematic drawing showing locations of tissues assayed relative to the site of ulceration: limbal conjunctiva adjacent to ulcer (A), conjunctiva 7mm from corneoscleral limbus (B), and limbal conjunctiva 180° away from ulcer (C).

In Case 1 tissues assayed were taken at two sites proximal to the ulcer, one sample at the limit of the ulcer, and one sample 7 mm distal from the ulcer (Fig. 7). Control (normal) tissue was taken from a site 180 degrees opposite the ulceration. Positive collagenolytic activity was seen on the two wells containing tissue proximal to the site of ulceration. No collagenolytic activity was seen for the wells containing balanced salt solution or normal tissue plus balanced salt solution.

In Case 2 a positive collagenolytic result was evident on the well containing tissue taken proximal to the ulcer (Fig. 8). The other wells contained balanced salt solution, normal tissue, and tissue distal to the ulcer. None of these gave evidence of collagen digestion.

DISCUSSION

Aronson and associates[7] proposed that peripheral corneal ulcers were the result

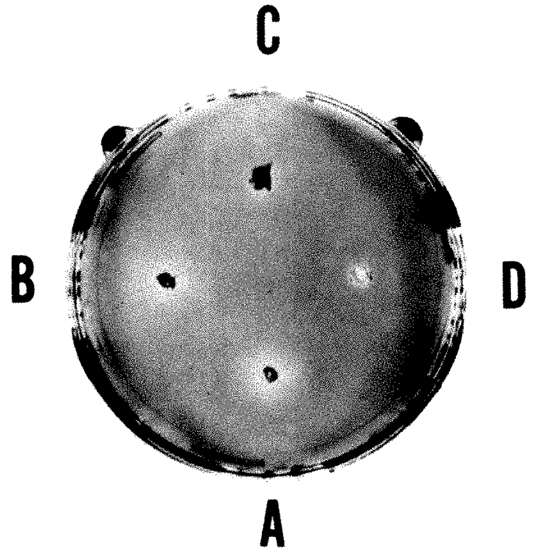

Fig. 7. (Eiferman, Carothers, and Yankeelov). Case 1. Results of radial diffusion assays performed. The letters indicate the locations of tissue samples relative to the ulcer (see Figure 6). Positive results, indicating collagenolytic activity, are seen on wells A and B, conjunctiva proximal to the ulcer. C indicates normal tissue taken 180 degrees opposite the ulcer; D indicates balanced salt solution.

of ischemic necrosis and advocated subconjunctival heparin therapy. Heparin therapy is not often successful. Because the cornea is 70% Type I collagen suggests that collagenolytic enzymes are a more reasonable explanation as the cause of ulceration.

The first animal collagenase was identified in tadpole tissue by Gross and Lapiere.[9] Since then, collagenase has been identified in over 30 animal tissue sources.[10] Slansky and associates[11] reported collagenase activity in corneal epithelium associated with acute or chronic corneal ulceration. Bloomfield and Brown[12] found that collagenase can be produced by both normal and inflamed rabbit conjunctiva. Additionally, Brown[13] reported that a collagenase and proteoglycanase are released from limbal conjunctiva in Mooren's ulcer. The role of collagenase in alkali burns has been well documented.[14-19]

Aviel[20] was the first to recognize that conjunctiva might be implicated in peripheral corneal disease. He successfully treated five cases of Mooren's ulcer with peritomy and cryotherapy. In addition to treatment of Mooren's ulcers,[4] conjunctivectomy has been used in treatment of rheumatoid thinning, rosacea keratitis, and thermal burns.[3]

Our study implicates the presence of collagenase in the limbal conjunctiva adjacent to marginal ulcers in rheumatoid patients. Beneficial results of surgical excision of conjunctiva adjacent to ulcers may operate through one or both of the following mechanisms: (1) the procedure may simply stimulate a supply of natural serum antiproteases such as α_2-macroglobulin via bleeding in the area; (2) the procedure may eliminate the proximity of conjunctiva and cornea or corneal epithelium. Other studies on culture of wounded tissue suggest that proximity of epithe-

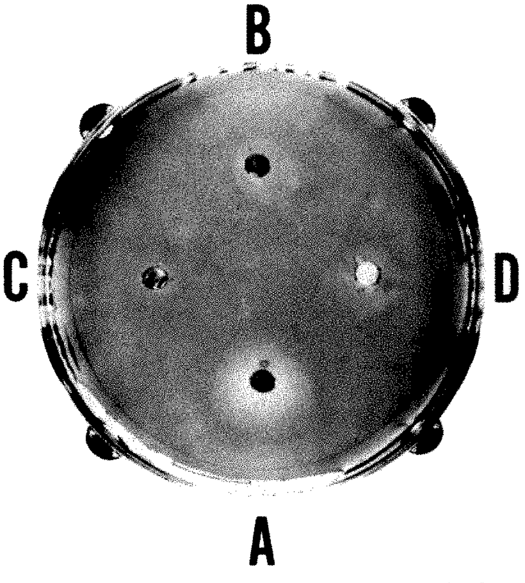

Fig. 8 (Eiferman, Carothers, and Yankeelov) Case 2. Results of radial diffusion assays. The letters indicate the locations of conjunctival samples relative to the ulceration (see Figure 6). Positive results are seen on well A; tissue taken proximal to the site of ulceration. B indicates conjunctiva taken 7 mm from the corneoscleral limbus; C indicates normal conjunctiva 180 degrees opposite the ulcer; D indicates balanced salt solution.

lium and mesenchymal tissue[21] enhance the production of collagenase.

Recent studies[22] suggest that topically applied α_2-macroglobulin may be of limited value in the control of corneal ulceration because of the inability of this inhibitor to penetrate the afflicted region. Whether or not α_2-M has a significant role in healing, conjunctiva adjacent to the ulceration will definitely release a proteolytic enzyme in concentrations capable of digesting lathyritic collagen.

Under the same conditions of incubation, normal conjunctiva reveals no evidence of collagenase production. Conjunctiva removed distal to the site of ulceration in the same eye did not produce a zone of digestion in the collagen-agarose gel. This suggests that the conjunctiva adjacent to the ulcerating cornea has been activated in some way to produce degradative enzymes. This activation may be initiated by some aspect of autoimmunity.

Thus, patients with Mooren's ulcer have circulating humoral antibody to corneal tissue[23] and these patients may possibly have an autoimmune basis for their disease. Similarly, many rheumatoid patients have antinuclear antibodies, positive rheumatoid factors, and antisalivary duct and antilacrimal gland antibodies.[24,25]

Circulating anticonjunctival and anticorneal antibodies were not found in either of our patients. The significance of the band of IgG along the basement membrane of the conjunctiva is not clear. This appears to be an indication of autoimmune phenomena occurring in the conjunctiva. Whether this is a cause or effect of the disease is unknown.

The inflammatory stage in wound repair[26] may have its counterpart initiated in rheumatoid ulceration as tissue damage elicited by humoral antibody. Some aspects of an inflammatory response may be induced on the periphery of the cornea and may trigger enhanced collagenase production in the conjunctiva and result in corneal ulceration. Consistent with this hypothesis is the observation that central wounds or burns of the cornea do not exhibit the more severe and prolonged ulceration characteristics of corneal wounds involving the corneoscleral limbus. This hypothesis, although attractive, leaves unanswered questions such as why do ulcers routinely appear inferiorly rather than superiorly or uniformly around the periphery of the cornea.

SUMMARY

Two patients with rheumatoid arthritis and peripheral corneal ulcerations were successfully treated by conjunctival resection. The tissues removed were assayed by a variation of the radial diffusion method for tissue collagenases. We used an agarose matrix containing lathyritic rat skin collagen. Wells 3 mm deep were punched in the agarose-collagen and surgical specimens were placed in the wells. Spaces remaining in the wells were filled with balanced salt solution. The assay dishes were incubated for four days at 32°C near 100% humidity. Under these conditions release of collagenase was detected by the clearing of diffuse zones in the gel surrounding the well. Conjunctiva proximal to the ulcer produced definite zones of lysis, whereas control specimens taken remote to the ulceration produced no lysis. This direct evidence for collagenase involvement offers an exploration for the beneficial effects of conjunctival resection.

ACKNOWLEDGMENT

Mr. Michael E. Bowles and Mr. Benjamin Van Osdol prepared the lathyritic collagen used in this study.

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COMPARISON OF THERAPEUTIC ROUTES IN EXPERIMENTAL *PSEUDOMONAS* KERATITIS

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Subconjunctival injection of antibiotics has been used for many years in the treatment of bacterial keratitis. Objective proof of the effectiveness of antibiotics by the subconjunctival injection route is lacking both in experimental and human infections.[1-3] Little is known about the effectiveness of antibiotics by the intramuscular route.[2,3]

We devised an experimental model of *Pseudomonas* keratitis suitable for objective evaluation of therapy.[4,5] Results were assessed quantitatively by determining the number of bacteria surviving in the cornea.[5] We report herein results of studies comparing the effectiveness of antibiotic administered by subconjunctival injection, intramuscular, or topical routes in the treatment of experimental *Pseudomonas* keratitis in guinea pigs and rabbits.

MATERIAL AND METHODS

Pseudomonas aeruginosa strains 107 and 113 were isolated from human corneal ulcers and were maintained by routine bacteriologic methods.[6] The strains were susceptible by in vitro tests to tobramycin.[5] Tobramycin was supplied in vials containing solutions of 40 mg/ml. Dilution of antibiotic for topical application were made with sterile 0.15M NaCl.

Male Hartley-strain guinea pigs weighing approximately 300 g and New Zealand white rabbits weighing approximately 2 to 3 kg were used in this study. Animals were anesthetised and were then infected intracorneally with about ten viable bacteria in a volume of 20 μl with a 30-gauge needle and microsyringe. Treatments were begun 24 hours later when all animals had frank keratitis. All topical therapy consisted of two drops every 30 minutes. Animals were killed either six or 24 hours after the beginning of treatment. Corneas were removed and ground in broth. Quantitative subcultures were made from serial dilutions in broth. Results were expressed as numbers of viable bacteria in the cornea in common logarithms (base 10). Results were evaluated statistically by analysis of variance.[7] Concentrations of tobramycin in serum were assayed with a modification of an enzymatic technique.[8]

For the therapy trials, all animals were infected bilaterally. Subconjunctival injections of antibiotic were made only in the right eyes. The right eyes were referred to as the ipsilateral eyes, and the left uninjected eyes were identified as the contralateral eyes.

RESULTS

Pharmacokinetic studies with intramuscular injection of tobramycin indicated that 1 mg/kg of body weight was the appropriate parenteral dose for rabbits. Absorption of tobramycin by the subconjunctival injection route was more rapid than by the intramuscular route but was less rapid than by the intravenous route (Table 1).

Serum concentrations after administration of 20 mg of tobramycin (approxi-

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TABLE 1

SERUM LEVELS OF TOBRAMYCIN IN RABBITS AFTER ADMINISTRATION OF 1 MG/KG OF BODY WEIGHT, BY INTRAMUSCULAR, INTRAVENOUS, AND SUBCONJUNCTIVAL ROUTES

Time (hrs)	Intramuscular (No.=5) (μg/ml) $\bar{x}$ ± S.D.	Intravenous (No.=5) (μg/ml) $\bar{x}$ ± S.D.	Subconjunctival Injection (No.=4) (μg/ml) $\bar{x}$ ± S.D.
1/4	6.3±2.3	4.5±0.8	3.6±1.5
1/2	Not done	3.2±0.3	5.0±1.3
1	6.5±1.2	2.1±0.4	4.0±0.6
2	3.6±0.9	1.9±1.2	2.4±0.7
3	2.3±1.4	0.2±0.2	1.8±0.2
4	1.1±0.7	None	1.2±0.7

mately 7 to 10 mg/kg of body weight) by the subconjunctival injection route were also determined (Table 2). The mean 30-minute concentration was approximately five times the serum concentration achieved after 1 mg/kg of body weight by the subconjunctival injection route.

The efficacy of tobramycin by the intramuscular and subconjunctival injection routes in combination with topical therapy was determined in guinea pigs (Table 3). Previous studies indicated that 2 mg/kg of body weight of tobramycin injected intramuscularly in guinea pigs achieved appropriate blood levels.[5] In this trial, topical therapy was effective alone. Neither intramuscular nor subconjunctival injection administration of antibiotic significantly enhanced the effectiveness of topical antibiotic during a six-hour trial.

In the next study, the efficacy of tobramycin, 2 mg/kg of body weight, by subconjunctival injection or intramuscular administration alone without topical therapy was evaluated (Table 4). All groups treated with antibiotic had significantly lower corneal colony counts than the group treated with saline alone ($P < .001$). The effectiveness of one or two doses of antibiotic by the intramuscular route was not significantly different from that of one dose of antibiotic by the subconjunctival injection route. Two doses of tobramycin by the subconjunctival injection route did achieve a greater therapeutic effect in the ipsilateral eyes than in the contralateral eyes or in the other treatment groups.

TABLE 2

SERUM CONCENTRATIONS IN THREE RABBITS AFTER SUBCONJUNCTIVAL INJECTION OF 20 MG OF TOBRAMYCIN

Time (min)	Serum Concentrations (μg/ml) $\bar{x}$ ± S.D.
30	24.5 ± 0.3
60	21.7 ± 1.0
120	10.3 ± 3.1
180	6.2 ± 1.3

The efficacy of tobramycin by topical, intramuscular, and subconjunctival injection routes was determined in rabbits with experimental *Pseudomonas* keratitis (Table 5). Tobramycin, 1 mg/kg of body weight, by the intramuscular or subconjunctival injection route was not significantly more effective than topical saline. Topical tobramycin, 40 mg/ml, was effective.

Tobramycin, 20 mg, by the subconjunctival injection route (Table 6) had a significant therapeutic effect in the ipsilateral injected eyes as compared to the saline treatment group, the intramuscular treatment group, or the left contralateral eyes ($P = .002$). However, tobramycin, 20 mg/ml, by the topical route was more effective than tobramycin, 20 mg, by the subconjunctival injection route.

TABLE 3

SIX-HOUR THERAPY TRIAL WITH TOBRAMYCIN BY TOPICAL, INTRAMUSCULAR, AND SUBCONJUNCTIVAL INJECTION ROUTES IN EXPERIMENTAL KERATITIS IN PIGS WITH *PSEUDOMONAS* 107*

Treatment	Corneal Colony Counts $\bar{X}$ ± S.D.	 (No.)		P
0.15M NaCl, topical and one subconjunctival injection dose at 0 hr	5.80 ± 0.45	(6)		
Tobramycin, 20 mg/ml				
Topical	3.90 ± 0.58	(6)		
Topical and 2 mg/kg of body weight, intramuscular, at 0 hr	3.42 ± 0.79	(6)		< .001
Topical and 2 mg/kg of body weight, subconjunctival injection, at 0 hr, right eye			.61	
Right eyes	3.41 ± 0.36	(6)		
Left eyes	3.64 ± 1.15	(6)		

*Topical therapy consisted of two drops every 30 minutes.

TABLE 4

SIX-HOUR THERAPY TRIAL WITH TOBRAMYCIN, 2 MG/KG OF BODY WEIGHT, BY INTRAMUSCULAR AND SUBCONJUNCTIVAL INJECTION ROUTES IN EXPERIMENTAL KERATITIS IN GUINEA PIGS WITH *PSEUDOMONAS* 107

Treatment	Corneal Colony Counts $\bar{X}$ ± S.D.	 (No.)	P
0.15M NaCl, subconjunctival injection, 0 and 2 hrs	6.26 ± 0.27	(6)	
Tobramycin intramuscular injection,			
0 hr	5.69 ± 0.24	(6)	
0 and 2 hrs	5.45 ± 0.49	(6)	.32
Tobramycin subconjunctival injection, 0 hr, right eye			
Right eyes	5.50 ± 0.39	(6)	
Left eyes	5.89 ± 0.42	(6)	
Tobramycin subconjunctival injection, 0 and 2 hrs, right eye			
Right eyes	4.73 ± 0.60	(6)	
Left eyes	5.51 ± 0.58	(6)	.005

TABLE 5

SIX-HOUR THERAPY TRIAL WITH TOBRAMYCIN BY INTRAMUSCULAR, SUBCONJUNCTIVAL INJECTION, AND TOPICAL ROUTES IN EXPERIMENTAL KERATITIS IN RABBITS WITH *PSEUDOMONAS* 107*

Treatment	Corneal Colony Counts $\bar{X}$ ± S.D.	 (No.)		P
0.15M NaCl, topical	7.04 ± 0.44	(4)		
Tobramycin, 1 mg/kg of body weight, intramuscular, 0 hr	7.02 ± 0.22	(6)		
Tobramycin, 1 mg/kg of body weight, subconjunctival injection, 0 hr, right eye			.92	
Right eyes	6.71 ± 0.35	(5)		
Left eyes	7.03 ± 0.22	(5)		< .001
Tobramycin, 40 mg/ml, topical	3.32 ± 1.75	(6)		

*Topical therapy consisted of two drops every 30 minutes.

TABLE 6

SIX-HOUR THERAPY TRIAL WITH TOBRAMYCIN BY TOPICAL, INTRAMUSCULAR, OR SUBCONJUNCTIVAL INJECTION ROUTES IN EXPERIMENTAL KERATITIS IN RABBITS WITH *PSEUDOMONAS* 107*

Treatment	Corneal Colony Counts $\bar{x}$ ± S.D.	(No.)	P	
0.15M NaCl, topical	7.25 ± 0.29	(6)		
Tobramycin, 20 mg, intramuscular, at 0 hr	6.94 ± 0.26	(6)		
Tobramycin, 20 mg, subconjunctival injection, at 0 hr, right eye			.007	
Right eyes	5.48 ± 1.50	(6)		
Left eyes	6.91 ± 0.19	(6)		< .005
Tobramycin, 20 mg/ml, topical	3.93 ± 1.01	(6)		

*Topical therapy consisted of two drops every 30 minutes.

The numbers of bacteria in the eyes of the group treated with 20 mg of tobramycin intramuscularly or in the left, contralateral eyes of the subconjunctival injection treatment group were not significantly different from those of the saline group ($P = .74$). Thus, there was no systemic therapeutic effect of this dose by either intramuscular or subconjunctival injection routes.

The next trial was designed to determine whether antibiotic by the subconjunctival injection route would improve the effectiveness of topical therapy in the rabbit (Table 7). Tobramycin, 20 mg, by the subconjunctival injection route did not significantly enhance the effectiveness of 20 mg/ml of topical tobramycin.

In a trial with *Pseudomonas* strain 113, the effectiveness of topical tobramycin was compared with that of tobramycin by the subconjunctival injection route in rabbits (Table 8). Tobramycin by the subconjunctival injection route was not significantly more effective than topical saline. Topical tobramycin, 20 mg/ml, was effective.

A 24-hour trial was carried out comparing topical therapy alone with a single dose by the subconjunctival injection route at the beginning of therapy (Table 9). Numbers of bacteria in the left eyes of the subconjunctival injection group were not different from the saline-treatment group ($P = .79$). Antibiotic by the subconjunctival injection route was more ef-

TABLE 7

SIX-HOUR THERAPY TRIAL WITH TOBRAMYCIN BY TOPICAL AND SUBCONJUNCTIVAL INJECTION ROUTES IN EXPERIMENTAL KERATITIS IN RABBITS WITH *PSEUDOMONAS* 107*

Treatment	Corneal Colony Counts $\bar{x}$ ± S.D.	(No.)	P	
0.15M NaCl topical	7.18 ± 0.14	(6)		
Tobramycin, 20 mg/ml, topical	3.41 ± 1.75	(6)		
Tobramycin 20, mg/ml, topical and tobramycin 20 mg, subconjunctival injection, right eye			.29	< .001
Right eyes	3.21 ± 1.27	(5)		
Left eyes	4.24 ± 0.76	(6)		

*Topical therapy consisted of two drops every 30 minutes.

TABLE 8

SIX-HOUR THERAPY TRIAL WITH TOBRAMYCIN BY SUBCONJUNCTIVAL INJECTION AND TOPICAL ROUTES IN EXPERIMENTAL KERATITIS IN RABBITS WITH *PSEUDOMONAS* 113*

Treatment	Corneal Colony Counts $\bar{X} \pm$ S.D.	Corneal Colony Counts (No.)	P	P
0.15M NaCl, topical	7.06 ± 0.09	(6)		
Tobramycin, 20 mg, subconjunctival injection, right eye				
Right eyes	6.49 ± 0.63	(6)		
Left eyes	6.69 ± 0.23	(6)	.48	< .001
Tobramycin, 20 mg/ml, topical	3.37 ± 1.48	(6)		

*Topical therapy consisted of two drops every 30 minutes.

fective than saline but was much less effective than by the topical route.

In the final study, (Table 10), the efficacy of a single dose of tobramycin 20 mg by the subconjunctival injection route alone was determined by comparing results in right and left eyes.

DISCUSSION

In experimental *Pseudomonas* keratitis in guinea pigs and rabbits, topical therapy was consistently superior to administration of antibiotic by the subconjunctival injection or intramuscular routes. In one comparison in guinea pigs (Table 4) and four comparisons in rabbits (Tables 5, 6, 8, and 9) topical therapy was more effective than therapy by the intramuscular or subconjunctival injection routes. In two other trials, therapy by the intramuscular or subconjunctival injection routes did not improve efficacy of topical therapy (Tables 3 and 7). Comparable results were obtained both in guinea pigs and in rabbits.

Because the properties in vitro and in vivo are so similar, we expect that other aminoglycosides would also be more effective by the topical route than by the intramuscular or subconjunctival injection routes in this experimental model.

Data presented in Table 1 show more rapid absorption of antibiotic into the blood after subconjunctival injection administration than after intramuscular administration. We previously observed similar results in guinea pigs.[5] This observation may be because the periorbital tissues were very vascular.

One way to estimate the degree of effi-

TABLE 9

TWENTY-FOUR HOUR THERAPY TRIAL WITH TOBRAMYCIN BY TOPICAL AND SUBCONJUNCTIVAL INJECTION ROUTES IN EXPERIMENTAL KERATITIS IN RABBITS WITH *PSEUDOMONAS* 107*

Treatment	Corneal Colony Counts $\bar{X} \pm$ S.D.	Corneal Colony Counts (No.)	P	P
0.15M NaCl, topical	6.74 ± 0.31	(6)		
Tobramycin, 20 mg/ml, topical	0.79 ± 0.94	(6)		
Tobramycin, 20 mg, subconjunctival injection, at 0 hr, right eye				< .001
Right eyes	5.48 ± 1.08	(6)	.03	
Left eyes	6.60 ± 0.79	(6)		

*Topical therapy was given as two drops every 30 minutes.

TABLE 10

TWENTY-FOUR HOUR THERAPY TRIAL OF 20 MG OF TOBRAMYCIN BY THE SUBCONJUNCTIVAL INJECTION ROUTE ALONE IN EXPERIMENTAL KERATITIS IN RABBITS WITH *PSEUDOMONAS* 107*

Treatment	Corneal Colony Counts $\bar{X} \pm$ S.D.	Corneal Colony Counts (No.)	P
Tobramycin, 20 mg, subconjunctival injection, right eye			
Right eyes	4.78 ± 1.94	(6)	.06
Left eyes	6.55 ± 0.50	(6)	

*A single dose of tobramycin was injected in the right eye 24 hours after infection was established. No other therapy was given. Animals were killed 24 hours later.

cacy of 20 mg of tobramycin by the subconjunctival injection route was to compare numbers of bacteria in the right eyes that were injected with those of the left eyes which were not. In one trial (Table 6), the difference was highly significant (P = .002). In two other trials with the same organism (Tables 9 and 10), the difference was of marginal significance (P = .03; P = .06). In a trial with another strain (Table 8), there was no difference (P = .63). Topical therapy consistently had greater efficacy.

We have previously shown in guinea pigs that therapy by the intramuscular route had less variability of therapeutic effect than therapy by the topical route.[5] Specifically, the standard deviations of the means were smaller for intramuscular treatment groups than for topical treatment groups. The greater variability for the topical treatment groups is probably related to variables that are difficult to control, such as the interval between application of a drop and a blink. In our studies in which therapy by the subconjunctival injection route was effective, the standard deviations were large (Tables 6, 9, and 10). We do not believe the variability is related to the techniques of the injections.

Our results directly conflict with many reports that subconjunctival therapy yields high intraocular concentrations of drug.[1-3] Some differences may be the result of the method used. Baum and associates[9] reported that concentrations of gentamicin in the rabbit cornea were consistently higher with administration by the subconjunctival injection route than with the topical route in rabbits with *Pseudomonas* ulcers. However, these investigators compared topical therapy with 3 mg/ml of gentamicin, one drop every 30 minutes, with subconjunctival injection of 20 mg of gentamicin. In contrast, we compared 20 mg of tobramycin by the subconjunctival injection route with topical tobramycin, 20 mg/ml, two drops every 30 minutes. This higher concentration was used topically because it was more effective.[10] Because of differences in design and method, it was difficult to directly compare our results with those of Baum and associates.[9]

We are not aware of any studies in vivo that provide objective quantitative proof of the effectiveness of administration of antibiotics by the subconjunctival injection route.

Rabbits and guinea pigs with experimental *Pseudomonas* keratitis differ in response to parenteral antibiotic therapy. We previously showed in guinea pigs that intramuscular therapy with carbenicillin, gentamicin, or tobramycin was more effective than intramuscular saline.[5] In this study, administration of tobramycin by the subconjunctival injection route in guinea pigs was effective in the contralateral, uninjected eye as well as in the ipsilateral injected eye (Table 4). The doses of antibiotics used in these trials achieve appropriate blood levels.[5]

In sharp contrast, intramuscular therapy with tobramycin in rabbits was not more effective than saline (Tables 5 and 6). Furthermore, 20 mg of tobramycin by subconjunctival injection, which achieves high blood levels, did not have a therapeutic effect in the contralateral, uninjected eye (Tables 6, 8, and 9). The physiologic or anatomic mechanisms that account for this difference in response to therapy are unknown. Whether infection in the human cornea would more clearly resemble the guinea pig or rabbit is uncertain.

SUMMARY

We determined the efficacy of tobramycin administered by topical, intramuscular, and subconjunctival routes in guinea pigs and rabbits with experimental *Pseudomonas* keratitis. The topical route of administration was consistently more effective than either subconjunctival or intramuscular routes. Subconjunctival injection of antibiotic did not enhance the effectiveness of topical therapy in either guinea pigs or rabbits.

Intramuscular tobramycin was more effective than saline in guinea pigs with keratitis but not in rabbits with keratitis.

ACKNOWLEDGMENT

Tobramycin was supplied by Eli Lilly and Co.

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NOTES, CASES AND INSTRUMENTS

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To take advantage of the Zeiss camera system, we designed a platform to support infants or small animals while photographing the anterior segment or fundus (Figs. 1 and 2), or during fluorescein angiography.

The platform is a 15 × 30-inch rectangle of white lucite plastic positioned at a level 18.75 inches from the base of the camera table. It is mounted in a rigid frame of reinforced aluminum attached to two pins that fit into the receptacle normally used to support the chin rest of the Zeiss fundus camera. The platform is inset four inches from the lower vertical support to provide adequate working distance and full use of the joystick controls.

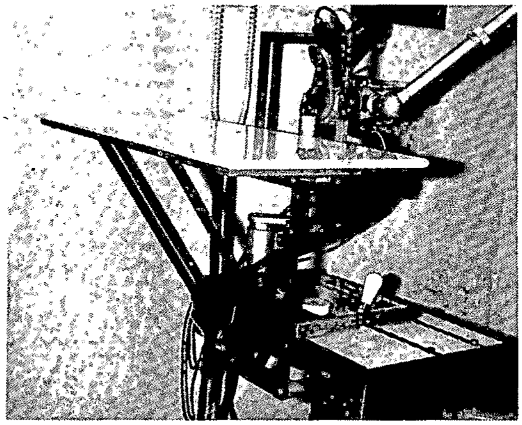

Fig. 1 (Foxworth and Nisbet). Pediatric photography platform attached to Zeiss Photo Slit Lamp.

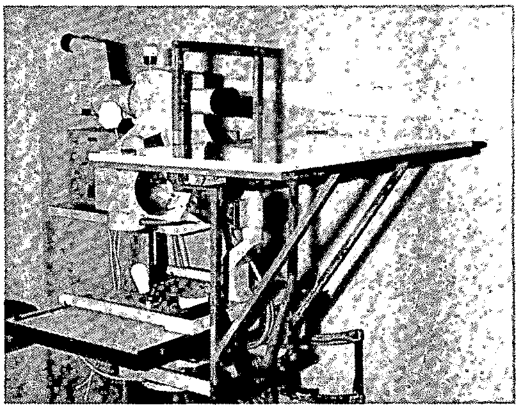

Fig. 2 (Foxworth and Nisbet). Pediatric photography platform attached to Zeiss fundus camera.

SUMMARY

We designed a rigid support platform to facilitate ocular photography of infants or small animals. The platform is versatile because it is compatible with either the Zeiss fundus camera or Zeiss Photo Slit Lamp.

BACILLUS LICHENIFORMIS CORNEAL ULCER

KHALID F. TABBARA, M.D.,

AND

NABIL TARABAY, M.D.
Beirut, Lebanon

Bacillus organisms, except for *B. anthracis*, are generally regarded as sapro-

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From the Department of Ophthalmology, American University Medical Center, Beirut, Lebanon. This study was supported in part by Grant No. 18-5116 from the School of Medicine, American University of Beirut, Beirut, Lebanon.

Reprint requests to Khalid F. Tabbara, M.D., Francis I. Proctor Foundation, 315-S, University of California, San Francisco, CA 94143.

phytes but can become pathogenic if the host's local or systemic defense mechanisms are altered. A number of species of the genus *Bacillus* have caused various ocular infections but rarely cause corneal ulcers. We describe herein a case of corneal ulcer caused by *B. licheniformis.*

CASE REPORT

A 46-year-old woman had pain, redness, and swelling of the right eye for 20 days. She said that the eye had been slightly injured by a twig two days before the onset of her symptoms.

On examination visual acuity was light perception in the right eye. Moderate swelling of the right upper eyelid was evident, the bulbar conjunctiva was injected and edematous, and there was a subconjunctival hemorrhage inferiorly. The cornea had a central ulcer with marked corneal necrosis and infiltration, and a 2-mm zone of clear cornea superiorly (Fig. 1). The anterior chamber contained a fibrin clot. The lens and fundus could not be visualized. The left eye showed no abnormalities. X-ray of the right orbit showed no evidence of an intraocular foreign body.

Scrapings from the ulcer, gram- and Giemsa-stained, contained gram-positive rods. Cultures of the scrapings showed a heavy growth of aerobic, spore-forming, gram-positive bacilli that were identified, on the basis of their biochemical reactions, as *B. licheniformis*[1] Table). The organism was sensitive to gentamicin sulfate, tetracycline, lincomycin hydrochloride, cephalothin sodium, cephradin, rifamycin, and trimethoprimsulfamethoxazole; it was resistant to penicillin G, erythromicin, ampicillin, and methicillin sodium.

The patient was given subconjunctival injections of gentamicin and staphcillin. After the antibiogram, we started a regimen of topical, subconjunctival, and intramuscular gentamicin sulfate. Improvement began on day 5; three weeks later the corneal ulcer had resolved and healing was underway (Fig. 2).

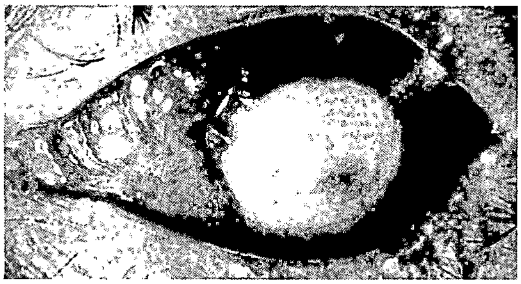

Fig. 1 (Tabbara and Tarabay). Extensive necrosis of the cornea in a patient with *B. licheniformis* corneal ulcer.

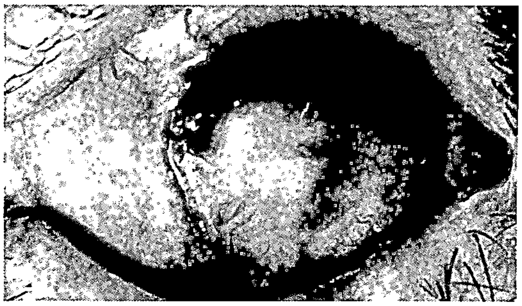

Fig. 2 (Tabbara and Tarabay). The same eye three weeks after treatment was begun.

DISCUSSION

Organisms of the genus *Bacillus* are aerobic, spore-forming, gram-positive rods. They are found commonly in air, soil, dust, hay, milk, water, and wool. With the exception of *B. anthracis*, they are usually nonpathogenic for man; however, exceptions exist. They have caused systemic diseases such as meningitis, pneumonia, and sepsis[2] (in both debilitated hosts and apparently normal hosts) and have been reported as causes of corneal ulcers[3] and endophthalmitis[4] after trauma or surgery. Because organisms of this genus are regarded as relatively insignificant; and their taxonomic relationships are complex and controversial, most clinical laboratories report any member of the *Bacillus* group as *B. subtilis* or "hay bacillus."

Several species of the *Bacillus* group recovered from eye infections have been identified as causative; for example, *B. laterosporus,*[5] *B. cereus,*[6] and *B. subtilis.*[7] Although *B. licheniformis* has been reported as a cause of sepsis in a patient with no known immunologic defect, to the best of our knowledge, the species has not previously been reported as a cause of ocular infection. Our patient had no debilitating disease or immune deficiency, but her injury may have disrupted the cornea's first line of defense, that is, its anatomic integrity. Minor corneal trau-

TABLE

CRITERIA FOR IDENTIFICATION OF *BACILLUS LICHENIFORMIS*

Test	Result
Gram stain	Long, thin, gram+, variably sized rod with subterminal spores
Growth at 37 and 50°C	Equal
Motility	Positive
Anaerobic growth	Positive
Hemolysis on rabbit blood agar	Beta hemolysis
10% lactose	Alkaline
Catalase	Positive
Oxidase	Positive
Salmonella-Shigella medium	Negative
Citrate	Positive
Urea	Negative
Nitrate	Positive
Indol	Negative
Methyl red	Negative
Voges-Proskauer	Positive
Triple sugar iron (TSI) agar (slant/butt)	Basic/Acid
Hydrogen sulfide production	Negative
Gelatin	Partially positive at 7 days
Litmus milk	Negative
Nutrient broth: 0% NaCl	Positive
6% NaCl	Positive
7% NaCl	Positive
Starch hydrolysis	Positive
Glucose	Acid
D-xylose	Acid
Mannitol	Acid and gas
Lactose	No change
Sucrose	Acid
Maltose	Acid

ma of this kind is believed to predispose to corneal infection.

SUMMARY

A 46-year-old woman developed a corneal ulcer after an injury to the right eye. Cultures were positive for *Bacillus licheniformis*. Although resistant to some antibiotics, the corneal ulcer resolved after treatment with topical, subconjunctival, and intramuscular gentamicin sulfate.

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BICONCAVE CONTACT LENS FOR VITREOUS SURGERY

JOHN G. SEBESTYEN, M. D.

Boston, Massachusetts

During trans pars plana vitreous surgical procedures adequate visualization of

---

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the fundus is essential. The posterior pole usually can be seen well with any of the customarily used plano-concave contact lenses. All of these lenses neutralize the refractive power of the cornea, thus making the retina accessible to viewing with the operating microscope. When the surgeon has to work at or near the equator of the globe, the eye has to be turned. This maneuver requires that the contact lens be tilted to a plane oblique to the surgeon's visual axis. The result will be that the surgeon sees the fundus through an oval, and therefore, smaller, pupil instead of a round one. At the same time, the more peripherally one has to look, the more tilting of the lens is required, and the more distorted the view becomes.

To minimize the need for turning the eye I designed a surgical contact lens* that has a much larger field of vision, which allows a view of the fundus as far as the equator. The lens is made of polymethylmethacrylate that has a refractive index of 1.49 by lathe cutting. It is biconcave in design (Figure), one of its surfaces has a radius of curvature of 12.5 mm, whereas the opposite surface is 13.5 mm. The diameter of the lens is 9.0 mm and its center thickness is 0.5 mm. Its refractive power is −75.50 diopters in air, by calculation.

The lens is applied to the cornea by floating it on balanced salt solution. Either the flatter or steeper surface faces the surgeon.

The image size is greatly reduced from that seen through a plano-concave lens. This difficulty is easily overcome by increasing the magnification of the operating microscope. Slight displacements of the lens from the central position bring into view the more peripheral parts of the fundus, because a strong optical lens has an induced prismatic power when a portion of the lens is used that is not in its optical axis. In the case of a −75.50 diopter lens, each millimeter away from the optical axis represents an approximately 7.5 prism diopter power displacing the image toward the center of the lens, that is, apex of the prism. The equator can be visualized easily by a minimal movement of the globe or by sliding of the lens. Lenses of any desired dioptric power and, therefore, different prismatic effects, can be designed by using the following formula

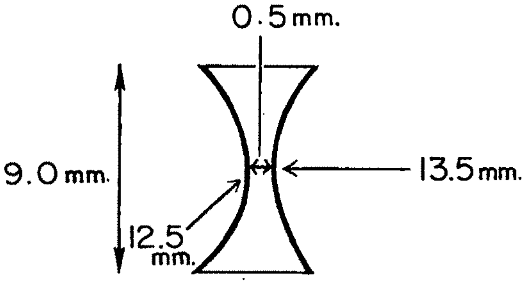

Figure (Sebestyen). Schematic drawing of biconcave contact lens.

$$D = \frac{1000\,(n - 1)}{r}$$

where *D* stands for dioptric power, n is the refractive index of the lens material, and r is the radius of curvature in millimeters.

SUMMARY

I designed a biconcave floating contact lens used for trans pars plana vitreous surgery. The lens uses the prismatic power of strong optical lenses to bring into view portions of the fundus that otherwise would be difficult to see.

---

*The lens was manufactured by LVR Contact Lens Laboratory, 636 Beacon St., Boston, MA 02215.

SCLERAL SPREADING FORCEPS FOR DRAINAGE OF SUBRETINAL FLUID

JERALD A. BOVINO, M.D.
Iowa City, Iowa

Drainage of subretinal fluid is one of the most critical and dangerous steps in retinal detachment surgery.[1] Little specialized instrumentation has been developed to aid the surgeon during this difficult task.

After a sclerotomy some surgeons grasp one scleral lip with toothed forceps to open the wound for drainage, which rotates the sclerotomy away from the surgeon and prevents optimal visualization of the choroid.

Cross-action capsule forceps have been modified, and eight semicircular opposed teeth 1 mm long have been added (Figure). The forceps can aid the surgeon at several stages during the drainage procedure:

(1) Frequently a few fine scleral fibers remain over the choroid after the sclerotomy incision has been completed. By using the forceps, these fibers can be put under tension to facilitate their incision.

(2) Use of the forceps facilitates inspection of the choroidal vasculature before perforation.

(3) The forceps allow the surgeon to spread the scleral lips by exerting symmetrical forces on the sclerotomy, and therefore the globe does not rotate in the orbit.

(4) The wound can be spread with one hand, allowing the surgeon to control the rapidity of the drainage.

I have used the forceps without complication during 20 retinal detachment procedures. The teeth are designed to interdigitate, as this facilitates insertion and removal, and minimizes the danger of choroidal puncture by the forceps. The instrument can be used for any size sclerotomy site, as the surgeon can vary the angle of approach and number of teeth inserted to fit the wound. If made of a nonmagnetic metal, the instrument could also be used for spreading a scleral incision over the pars plana, while extracting an intraocular foreign body by magnet.

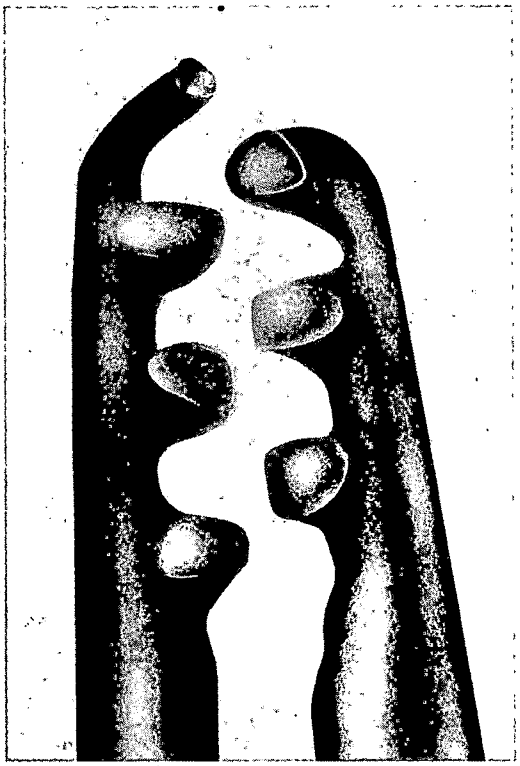

Figure (Bovino). The instrument consists of cross-action forceps 100 mm in length. It has a 90-mm straight section and a 10-mm angulated tip. Eight semicircular opposed teeth 1 mm long have been added to the tip.

SUMMARY

To faciliate drainage of subretinal fluid I modified cross-action forceps so that the 10-mm angulated tip had eight semicircular opposed teeth each 1 mm long.

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XII PAN AMERICAN CONGRESS OF OPHTHALMOLOGY

Some 1200 ophthalmologists representing each country in the Western hemisphere, together with others from Europe, attended the XII Pan American Congress of Ophthalmology in Miami, Florida, Feb. 25 to March 1, 1979. Edward W. D. Norton, president of the Congress, was aided by a large committee with notable contributions by the vice president, Richardo Martinez-Ferrer, and Don Nicholson, scientific director. It was the largest congress in the history of the Association. The traditional Pan American hospitality prevailed and the meeting was most successful.

The inaugural ceremonies opened in the grand ballroom of the Fontainebleau Hotel with an invocation by Bishop Augustin Roman of Miami. Professor Norton welcomed the visitors and was followed by the president of the Pan American Association Feliciano Palomino-Dena; Jules François of Ghent spoke for the Concilium Ophthalmologica Universal. Dr. Paul Rodriguez-Barrios of Uruguay gave the welcome for the Spanish speaking ophthalmologists; Clovis Paiva of Brazil for the Portuguese speaking ophthalmologists; and Bradley Straatsma of the United States for the English speaking ophthalmologists.

A. Edward Maumenee, who had attended the first meeting of the Pan American Association of Ophthalmology in Cleveland in 1940 and subsequently served as president of the association, was given the Gradle teaching medal in recognition of his extraordinary contribution to the success of the educational programs of the Pan American Association. The award was made by Professor Benjamin Boyd, executive secretary of the association who emphasized the support Dr. Maumenee had given him during his past

23 years managing the association. With Dr. Boyd's zeal and work as executive secretary-treasurer the association has emerged as a major educational, scientific, and cultural force in the Western hemisphere.

Robert Drews, who served the past 10 years as secretary-treasurer for North America, was awarded a certificate of appreciation. Dr. Guillermo Pico was named ophthalmologist of the Americas in recognition of the establishment and direction of the course he established in 1968 at the University of Puerto Rico. More than 230 Latin Americans and 69 Puerto Ricans have taken the 600-hour teaching course given annually from January to May in San Juan.

Robert N. Shaffer of San Francisco received the Pan American-Prevention of Blindness medal awarded jointly by the National Society for the Prevention of Blindness and the Pan American Association. Dr. Shaffer was one of the first ophthalmologists of the Americas to provide graduate teaching in the various countries of the Western hemisphere.

Dr.Norton paid particular tribute to Don Nicholson, scientific director of the program and to Ricardo Martinez-Ferrer, the vice president of the congress. The mayor of Miami then welcomed the guests. A huge reception followed in the adjoining ballroom where the ophthalmologists, their families, and associates renewed friendships and recalled earlier congresses and absent friends.

Before the opening of the meeting, the Pan American Society of Pathology convened for a three-day session. The Pan American Glaucoma Society met February 24th and the Pan American Society of Ophthalmic Surgery met February 25th. Then followed the most extensive scientific program in the history of the association, with symposia, scientific meetings, roundtables, instruction courses, and free papers; for example, in the first day of the meeting there were approximately 70 different papers, five scientific movies, and three roundtable sessions. Those sessions with simultaneous translation attracted large audiences, but attendance at other sessions waxed and waned depending upon the speaker and the topic.

The Pan American Association-American Journal of Ophthalmology lecture was given by Clement McCulloch of Canada, who spoke on the retinal pigmentary dystrophies with particular emphasis on choroidaremia, gyrate atrophy, and the different inheritance modes of various typed of retinitis pigmentosa. Dr. McCulloch was introduced by A. Edward Maumenee, President of the Ophthalmic Publishing Company.

Alberto Urrets-Zavalia, the Gradle lecturer, focused on the changes in retinal surgery with episcleral rather than intrascleral implants, cryotherapy rather than diathermy, and intraocular injection rather than subretinal drainage.

At the meeting of the council J. Wesley McKinney of the United States and Alfonso Gaitán Nieto were named delegates of honor in recognition of their long service to the association.

The first days of registration were difficult for the hotel and tourism committee, because the meeting was scheduled to open on Sunday, and many visitors arrived early to attend satellite meetings. Thus Pan Americans found that the Doral on the Beach Hotel was not honoring guaranteed advance paid reservations while at the same time accepting other guests. The Fontainebleau Hilton was overwhelmed early in the meeting, but Dr. Norton and his staff worked valiantly to obtain housing, even assigning unused hospital rooms.

Despite these disappointments the

meeting was first class in every respect. The level of ophthalmology in the Western hemisphere is rapidly rising. Ophthalmologists are making important and critical observations and the meeting reflected the remarkable technical advances of the past decade.

The XIII Congress is scheduled for Feb. 21-28, 1981, in Acapulco, Mexico.

FRANK W. NEWELL

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

A Maneuver to Elicit Vertical Dolls' Eye Movements

Editor:

In certain patients, such as those with Parkinson's disease or progressive supranuclear palsy, rigidity of the neck limits the ability to demonstrate vertical dolls' eye movements. The head resists being bent forward quickly enough to bring out passive contraversive eye movements. Daroff and Troost[1] wrote that "the oculocephalic responses are brisk in the horizontal direction, but nuchal rigidity usually limits vertical head movements." David, Mackey, and Smith[2] explicitly described in one of their patients with progressive supranuclear palsy "vertical movement of the neck required effort to overcome the rigidity and caused the patient to complain bitterly of pain."

We have had similar experiences and have found a simple maneuver to use in testing for vertical passive eye movements. The patient lies supinely on a stretcher that has wheels. The stretcher is sharply pushed either toward the feet or the head. The eyes can then be observed to move in a direction opposite to head movement (Figure). Occasionally the eye movements may be amplified by directing the patient to fixate a target flashlight or a particular spot on the ceiling.

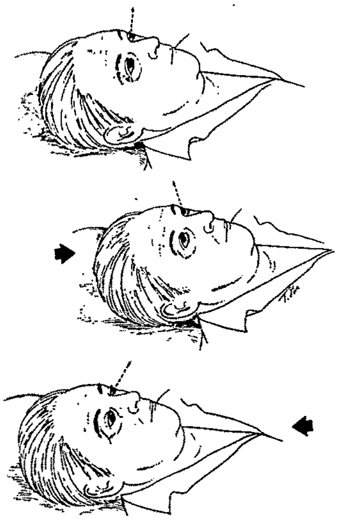

Figure. (Chu, Reingold, and Cogan). Top, While lying on a stretcher, the patient directs his eyes directly forward. Center, If the head of the stretcher is sharply pushed forward the eyes roll up. Bottom, If the foot of the stretcher is pushed back, the eyes roll down.

Compared to the gross body manipulations or passive head thrusts that are ordinarily used to determine the existence of vertical dolls' eye movements, our method has the advantages of simplicity for the examiner and comfort for the patient.

FRED C. CHU, M.D.
DOUGLAS REINGOLD, M.A.
DAVID G. COGAN, M.D.
Bethesda, Maryland

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The Mobile Eye Hospital

Editor:

In his editorial, "New initiatives in the war on blindness" (Am. J. Ophthalmol. 87:103, 1979), Alfred Sommer, celebrated the hopeful new step of the World Health Organization and the International Agency for the Prevention of Blindness toward a global attack on a shameful scourge in human existence, the continuing plight of those with curable or preventable blindness. The new initiatives confirm a strategy which uses the Mobile Eye Hospital developed in rural India over 30 years ago. The strategy is to combat blindness not only through education in preventive measures but also through treatment and cure.

The Mobile Eye Hospital was devised in direct response to existing realities. Most villages of developing countries still lack primary health care facilities and personnel. Even in developing countries with good facilities, many potential patients either are unaware of their availability or are unwilling to travel to one. The Mobile Eye Hospital brings modern eye service of the highest standards to such rural populations. The program and services are fully described in our monograph, "The Curable Blind: A Guide for Establishing and Maintaining Mobile Eye Hospitals" (Rambo and Chatterjee; Philadelphia; Rambo Committee for Curable Blind, Inc., 1974). After an advance public relations effort, a highly experienced and adequately equipped cadre of eye specialists and support personnel establish the Mobile Eye Hospital site at a locally provided school or temple. The work of the Mobile Eye Hospital team includes: screening and case finding, treatment (cataract extraction, general and plastic ophthalmic surgery, refraction, and optical service), consumer education (for example, nutrition and hygiene instruction for blindness prevention), provider education, and survey: remembering that all other aims are secondary to the aim of restoring sight to the curably blind. The Mobile Eye Hospital not only serves large groups of people economically with rapid delivery of ophthalmic services, but its success rates match or exceed those of modern Western hospitals.

The hope for sight offered to the curable blind by the Mobile Eye Hospital galvanizes local community interest and support that would otherwise not be forthcoming. Restoring sight to the curable blind appears to me not only the most humane but also the most effective strategy in establishing programs for nutrition and hygiene training to prevent blindness due to malnutrition and infection.

Bravo for the World Health Organization and the International Agency for the Prevention of Blindness in their new initiatives in the global war on blindness!

VICTOR C. RAMBO, M.D.
Philadelphia, Pennsylvania

Reply

Editor:

Removing the vast numbers of blinding cataracts afflicting inhabitants of developing countries is certainly an urgent priority of the war on blindness. The Mobile Eye Hospital scheme, pioneered by Dr. Rambo, has restored sight to countless villagers throughout India, Pakistan, and Bangladesh, and these efforts are rapidly being expanded.

In the past, all operations were carried out by fully trained ophthalmologists. Unfortunately, in many areas of the world there is a critical shortage of eye surgeons. Some African countries do not have even one eye surgeon. New initiatives are required to alleviate the problem: heavy use of highly trained auxillaries permits Dr. Christy and his colleague in Taxila, Pakistan, to perform over 10,000 cataract operations per year in their stationary village hospital. (Dr. Christy alone sometimes performs over 200 cataract operations a day.) Dr. Randy Whitfield and his colleagues are training highly selected "technicians" to remove cataracts in Kenya; and a program is underway to train general medical doctors in Bangladesh to staff rural cataract camps. In every instance it is clearly understood that the operations will be supervised, if not actually performed, by ophthalmic surgeons.

Whereas some might shudder at the thought of cataract surgery being performed by anyone less qualified that a fully trained ophthalmologist, something must be done to alleviate this enormous problem. Because of lack of sophisticated instruments, sutures and antibiotics, the primitive facilities in which the operations must be performed, and deficiencies in personal hygiene and presence of concommitant ocular diseases in these patients, the results will surely not match those of modern ophthalmic institutions. But with adequate training, supervision, and attention to details, cataract camps already achieve a success rate of 90 to 95%. If 90% of the millions presently blinded by cataracts can be rehabilitated the results will have been worth the price.

ALFRED SOMMER, M.D.
Bandung,Indonesia

Preoperative and Postoperative Visual Acuity

Editor:

When THE JOURNAL publishes articles describing the results of the various methods of cataract extraction, the authors usually furnish the postoperative visual acuity, but seldom give the preoperative visual acuity. This omission limits the usefulness of the information. I believe it would be scientifically more valid to know what change occurred in the visual acuity after the operation. I suggest that you institute an editorial policy in which authors submit both pre- and postoperative visual acuity.

IRVIN S. TAYLOR, M.D.
Scarsdale, New York

Risk: Benefit Ratio for Implant Surgery

Editor:

In the article, "Medicolegal hazards of intraocular lens implanting" (Am. J. Ophthalmol. 86:496, 1978), Jerome W. Bettman has succinctly pointed out the medicolegal hazards of intraocular lens implanting. With his vast knowledge in the medicolegal field, Dr. Bettman has written a timely article, one which outlines vital guidelines for every implant surgeon.

In the discussion of the risks:benefit ratio, he points out an area that presents a potential hazard to implant surgeons. To illustrate the principle, Dr. Bettman quotes from material published under my name in the American Intraocular Implant Society Journal 3:119,1977, in which 30 patients of the reported 103 were operated on with 6/18 (20/60) recorded visual acuity or better. Particularly well taken is the admonition "the anlage for litigation is present and, if the surgical result is not good, beware." However several factors regarding the original publication were not known to its readers. Because of misunderstanding, the material in the American Intraocular Implant Society Journal was printed without my knowledge or my approval. The statistics submitted at the request of the Implant Society were to be used, I believed, in conjunction with figures collected throughout the country to give a broad view of the status of lens implantation. They were published, however, as a completed study.

Had I submitted the material for publication: (1) It would have been accompanied by a narrative; (2) in addition to the visual acuity obtained in dim illumination, the visual acuity obtained under full illumination would have been included; (3) in addition to the distance visual acuity a statement of the near vision would have been essential; (4) I should have had the opportunity to proofread the material before it went to press; in the article under discussion a zero was dropped, changing the preoperative visual acuity in one patient from 20/200 to 20/20.

The question of when surgery is indicated plagues every surgeon. If, postsurgically, in surveying indications for surgery one considered only (1) the distance vision as opposed to both distance and near vision; and (2) only the results acquired in a dimly lighted room as opposed to results acquired in a brightly lighted room, many cases would fall to scrutiny. It is important to have access to all available material and to consider all factors before far-reaching conclusions are made.

JAMES H. GENTRY, M.D.
Denver, Colorado

Reply

Editor:

It is unfortunate that Dr. Gentry did not have an opportunity to approve of his article before it appeared in the American Intraocular Implant Society Journal. Had I known, I would not have used it as a reference. I could have used many other examples of the visual acuity having been relatively good before implantation of a pseudophakos.

JEROME W. BETTMAN, M.D.
Woodside, California

Use of Dilute Drug Solutions for Routine Cycloplegia and Mydriasis

Editor:

In the article, "Use of dilute drug solutions for routine cycloplegia and mydriasis" (Am. J. Ophthalmol. 86:820, 1978), by C. Brown and C. Hanna, the authors suggest that many patients usually receive multiple eyedrops, and that the effectiveness is reduced because of the irritation of the drops, which produces tearing and rapid washout.

It has long been my observation, during my own residency training and subsequently during my association with a second university training program, that patients being examined receive repeated applications of eyedrops. Overzealousness of ancillary personnel contributes; impatience of the examiner is also sometimes a factor.

In the order of my examination, I am ready to dilate the pupils after having performed biomicroscopy and applanation tonometry. It is quick and simple to administer one drop of tropicamide 1% to the anesthetized eyes before sending the patient out of the examining room. As mentioned by the authors, use of topical anesthetics eliminates or greatly reduces the irritating effect of the drops. It is also thought that topical anesthesia enhances corneal permeability and thus improves absorption of the drug. I rarely find it necessary to use more than one drop for routine examinations; I may add one drop of phenylephrine 10% to eyes with darkly pigmented irides.

In the absence of a commercially available way to administer microdrops of diluted solutions as reported by the authors, the above technique is effective, comfortable, and significantly cheaper than the apparently common practice of multiple applications. Tropicamide 1% costs $10.25 per 15-ml bottle.

JOHN B. H. CALDWELL, M.D.
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Reply

Editor:

We reported on the use of commercially available cycloplegic and mydriatic eye solutions given as microdrops (0.005 of 0.01 ml) or 10x diluted regular drops (about 0.07 ml). We are not aware of any commercially available apparatus to deliver microliter eyedrops; however, pharmacists could dilute the commercially available preparations. Dr. Caldwell's procedure of dilating the pupil after having used a topical anesthetic for applanation tonometry may be a common one and avoids multiple applications of eyedrops. It would be interesting to know if 0.1% tropicamide or 0.1% cyclopentolate would not be just as effective. The use of topical anesthetic followed by tonometry could increase drug absorption. The length of action of either of these drugs can be increased when more drug reaches the aqueous humor. However, the amount of drug in the aqueous humor necessary to produce mydriasis may be minute. In recent studies with Dr. Charles Thornton, we found that marked mydriasis was produced after the single application of 0.03 mg of pure phenylephrine hydrochloride powder. Unfortunately cyclopentolate and tropicamide powders are not available for human experimentation. Alani approached this problem in a different way by dipping the tip of a rod into 0.5% tropicamide solution.[1] When needed, the tip of the dried rod was touched to the conjunctiva. Attempts have been made in the past to suspend the cycloplegic and mydriatic drugs in ointment to the conjunctiva as a single application.[2,3] We hope that less irritating mydriatic and cycloplegic preparations will become available.

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REFERENCES

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2. Hendrickson, R. O., and Hanna, C.: Use of drugs in ointment for routine mydriasis. Ann. Ophthalmol. 9:333,1977.
3. Cable, M. K., Hendrickson, R. O., and Hanna, C.: Evaluation of drugs in ointment for mydriasis and cycloplegia. Arch. Ophthalmol. 96:84, 1978.

BOOK REVIEWS

Current Concepts in Cataract Surgery. Edited by Jared M. Emery. St. Louis, C. V. Mosby, 1978. Hardcover, 587 pages, index, preface, list of contributors, 524 black and white figures. $64.50.

This text containing the proceedings of the Fifth Biennial Cataract Surgical Congress is informal in style, full of interesting and usually humorous interchanges between the panelists. This may make for a good meeting but it does not often succeed in print. We all speak incoherently at times, and repetition and redundancy may be allowable in speaking to clarify a point. In a transcription of the conversation, however, one is frequently left only with the appreciation of a witticism rather than with an understanding of the subject.

This criticism does not apply to the papers themselves, which are brief and to the point. Most of them consist of the description of a technical maneuver, or procedure, or instrument that makes cataract surgery easier. As might be expected, a great deal of the text material is devoted to intraocular lenses and the argument goes on as to which is the best lens.

In all fairness I should mention several exceptions to the "here's how I do it" type of paper that is the bulk of the text. Perry Binder provided a good comparison of through and through suturing of corneal incisions and deeply placed suturing in the rabbit eye. At least in the rabbit the deeply placed sutures are preferable to through and through sutures.

A second precise piece of work was presented by Michael Callahan who performed endothelial cell counts after different kinds of cataract surgery. The cell loss varied but averaged about the same for all procedures.

The text contains few other purely scientific presentations. In contrast, a large amount of space is devoted to polls of the audience to establish some point or other. It is difficult to know exactly what is achieved by these oral expressions of opinions. This reviewer would like to point out that if a poll were taken by Columbus in 1491, most of his audience would have insisted the world was flat. Collective error does not make a truth.

DAVID SHOCH

Clinicopathologic Correlation of Ocular Disease. A Text and Stereoscopic Atlas, 2nd ed. By David J. Apple and Maurice F. Rabb. St. Louis, C. V. Mosby Co., 1978. Hardcover, 526 pages, 758 illustrations and 112 stereoscopic views in color. $110.

Understanding ocular pathology gives an additional dimension to the understanding of clinical ophthalmology. Thus, ocular pathology is not one of the subjects that an ophthalmologist may or may not choose to become involved with. Knowledge of ocular pathology is an intricate and essential part of practical ophthalmology. Without it, complete understanding of the nature, the course, the results, and the treatment of eye diseases is impossible. In many instances exact diagnosis, classification, and recognition of a cure in ocular disease are only possible with the help of pathologic study. Correlation of the pathologic changes with the clinical findings is the key for smooth interaction of the two aspects of ocular disease. The present book centers on this correlation in an outstanding way.

Why does the fact that clinical observations are made in living tissues with instruments such as the slit lamp and the ophthalmoscope, whereas pathologic findings are obtained in preserved tissues, result in separate teaching? This text has overcome this artificial separation.

In this new edition, one new chapter and many new illustrations have been added. The chapters are as follows: Introduction, Developmental anomalies, Cornea, Lens, Glaucoma, Uvea, Fundus diseases, Retinoblastoma Leukocoria and Phakomatoses, Optic nerve, and Conjunctiva and Eyelids. In these chapters the authors have combined all practically important clinical and pathologic knowledge of eye disease a well organized presentation, which is supported by outstanding illustrations and tables in the text as well as by colored stereoscopic views. The book also gives the normal anatomy and histology necessary for complete understanding. Extensive and well-selected references are offered at the end of each chapter for follow-up reading.

This book is a resident's dream come true. It brings together what never should have been separated in the first place. It makes learning and understanding easy in instances where this is difficult or impossible with the clinical or the pathologic approach alone. The book is well written and the authors have used good judgement and discipline in their selection of material.

How fortunate we are to have the new and improved edition of this book! It is most enthusiatically recommended to all who would like to be introduced to or who need to update their knowledge of ophthalmology seen as a whole from a clinicopathologic viewpoint.

J. REIMER WOLTER

SYMPOSIA

Strabismus. Edited by Robert D. Reinecke. New York, Grune and Stratton, 1978. Hardcover, 443 pages, index, table of contents, 168 black and white figures, 59 tables. $22.50

GUILLERY, R. W.: **Normal, deprived and congenitally abnormal geniculocortical pathways**

SHERMAN, S. MURRAY: **Development of the geniculocortical pathways**

VON NOORDEN, GUNTER K.: **A primate model for amblyopia**

AWAYA, SHINOBU: **Stimulus vision deprivation amblyopia in humans**

HUBER, A., AND MEYER, E.: **A new method of oculography**

METZ, HENRY S.: **Saccadic velocity studies with mechanical restriction of ocular mobility**

PIGASSOU-ALBOUY, RENEE, AND JONES, SAMUEL T.: **The cover test**

D'ESPOSITO, M., CENNAMO, F., CENNAMO, G., AND DE FRANCISCIS, P.: **Prototype of an ophthalmotrope**

STRACHAN, IAN M.: **Isometric force measurements from the horizontal muscles in exodeviations**

TSUTSUI, JUN, AND FUKAI, SAKUKO: **Human strabismic cases suggestive of asymmetric projection of the visual pathway**

RUBIN, WILLIAM, HELM, CARL, AND MCCORMAK, MICHAEL K.: **Ocular motor anomalies in monozygotic and dizygotic twins**

KATSUMI, OSAMU., AND UEMURA, YASUO: **The comparative study of the incidence of unilateral amblyopia during the past ten years**

ING, MALCOLM, R., AND PANG, STEPHANIE, W. L.: **The racial distribution of strabismus**

HARCOURT, BRIAN, MEIN, JOYCE, CROMPTON, JOHN, AND JOHNSTON, PHILEO. **The significance of retraction of the globe on infraduction in certain cases of thyroid eye disease.**

GREGERSEN, E., PONTOPPIDAN, J., AND RINDZIUNSKI, E.: **Intra and interindividual variation in atropine-resistant residual accommodation**

WEISS, J.B.: **Differential electro-oculography**

WATSON, P.G., BANKS, R.V., CAMPBELL, F.W., AND HESS, R.F.: **A clinical assessment of a new treatment for amblyopia**

VERIN, PHILLIPPE, VILDY, ALAIN, AND MAURIN, JEAN FRANÇOIS: **Dynamic visual acuity and nystagmus**

REINECKE, ROBERT D.: **Downbeat nystagmus and strabismus**

OHMI, EMIKO, FUJITA, JUNKO, TUBOI, TOSHIKO, OGLI, KENJI, AND KANI, KAZUTAKA: **Electrophysiological analysis of congenital nystagmus before and after Anderson's surgery**

BERARD, P.V., MOUILLAC, N., REYDY, R., AND SPIELMANN, A.: **Surgical treatment of congenital nystagmus with horizontal torticollis**

VIRDI, PREM SINGH: **Abduction paralysis and ocular deviation**

AUST, WOLFRAM: **The range of fusion in reduced monocular and binocular vision**

BAGOLINI, BRUNO: **Orthoptic and prismatic treatment of congenital nystagmus**

ISHIKAWA, SATOSHI: **Latent nystagmus and its etiology**

SCHLOSSMAN, ABRAHAM, AND MUCHNICK, RICHARD S.: **Nystagmus blockage: classification and surgical management**

MUHLENDYCK, H.: **The Faden operation in the treatment of congenital nystagmus**

BICAS, HARLEY E.A., FARIA-SOUSA, SIDNEY J., AND MIDORCIAVA, RUY: **Weak cycloplegia for correction of exodeviations Part I: rationale and selection of solutions based on depth of cycloplegia within two hours**

BICAS, HARLEY E.A., MIDORICAVA, RUY, AND FARAI-SOUSA, SIDNEY J.: **Weak cycloplegia for correction of exodeviations, Part 2. Duration of partial cycloplegia and values of the AC/A ratio.**

PRATT-JOHNSON, J.A., BARLOW, J.M., AND TILLSON, G.: **Early surgery in intermittent exotropia**

WONG, GUY Y., AND YOUNG, JEAN: **The tight lateral rectus syndromes**

HARDJOWIJOTO, SANJOTO: **Vertical microstrabismus with large latent deviation**

CAMPOS, EMILIO C., ENOCH, JAY M., AND RABINOWICZ, I. MATTHEW: **Prismatic therapy for unilateral aphakia due to congenital cataract**

CALDERIA, JORGE ALBERTO F.: **Basic steps in the recession of the superior oblique muscle**

DE DECKER, W., AND CONRAD, H.G.: **Rotating surgery in cases of severe head tilt**

ROSENBAUM, ARTHUR, METZ, HENRY, CARLSON, MEL, AND JAMPOLSKY, ARTHUR: **Indications for adjustable rectus recession**

SCOTT, ALAN B.: **Planning inferior oblique muscle surgery**

SPIELMANN, ANNETTE, AND LAULAN, JEAN: **Action of recessions and resections when associated with Cuppers fadenoperation in estropia: statistical results**

AICHMAIR, H.: **Further experiences with muscular neurotisation in surgery of traumatic abducens paresis**

FELLS, PETER, AND MARSH, RONALD J.: **Anterior segment ischaemia following surgery of two rectus muscles**

ALMEIDA, HENDERSON C.: **Complications of strabismus surgery**

WANATABE, YOSHIMASA, REIKO, MORI, AND OHTSUKI, HIROSHI: **Studies on the Bielschowsky head tilt test**

GUYTON, DAVID L., AND VON NOORDEN, GUNTER K.: **Sensory adaptions to cyclodeviations**

INATOMI, AKIHIRO, TERAO, NAOMICHI, AND OKAMOTO, KIYOKO: **Measurement of compensatory cycloduction by fundus photographic method**

VELEZ, GUILLERMO: **Calibrated measurement of eye movements in lateral rectus paralysis**

YAZAWA, KOJI, KATO, KAZUO, AND KAWAMURA, MIDORI: **Liquid crystal phase difference projection haploscope**

FRANCE, THOMAS, D., AND BURBANK, DANIEL P.: **A computerized model of human ocular movements**

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Annals of Internal Medicine

PROBABLE TRANSMISSION OF *TOXOPLASMA GONDII* BY ORGAN TRANSPLANTATION. Ryning, F. W., McLeod, R., Maddox, J. C., Hunt, S., and Remington, J. S. (Depts. Med. and Pathol., Stanford Univ. School of Med., Stanford, Calif.). Ann. Intern. Med. 90:47, 1979.

Two heart transplant recipients developed toxoplasmosis shortly after surgery. As neither recipient had serologic evidence of exposure to *Toxoplasma gondii* before transplantation, infection was acquired from an exogenous source. Of the possible modes of transmission of *Toxoplasma* to the recipients, infection by the oral route or through transfusion of blood or blood products seems remote. Since both heart donors had serologic evidence of recently acquired toxoplasma infection at the time of transplantation and the myocardium is known to be infected with the organism during acute infection, we considered these facts to be a strong implication that the donor's hearts were the most likely source of toxoplasma infection in the recipients. (1 table, 16 references)—Authors' abstract

Annals of Neurology

VISUAL EVOKED POTENTIALS AND THE VISUOGRAM IN MULTIPLE SCLEROSIS. Bodis-Wollner, I., Hendley, C. D., Mylin, L. H., and Thornton, J. (Depts. Neurol. and Biostatistics, Mount Sinai School of Med., New York, N.Y.), Ann. Neurol. 5:40, 1979.

Visual evoked potential (VEP) latency to a sinusoidal grating pattern was measured in each eye of 103 patients with multiple sclerosis (MS) and compared with results in a control group of 56 patients hospitalized for other neurological conditions. Of the 50 patients classified as having definite MS, 90% showed prolonged latency (over 131 msec) in one or both eyes. In each eye of 24 of the MS patients, psychophysical measurement of the detectability of grating patterns was obtained. This test was abnormal in 11 of 13 patients with definite MS, three of four with probable MS, and five of seven with possible MS. There was no concordance between prolonged VEP latency and visual impairment as revealed by the psychophysical test. Apparently pathways determining VEP latency and spatial contrast detection may be unequally affected in MS. (5 figures, 28 references)—Authors' abstract

Archives of Ophthalmology

CONGENITAL NYSTAGMUS SURGERY: A QUANTITATIVE EVALUATION OF THE EFFECTS. Dell'Osso, L. F., and Flynn, J. T. (Dept. Neurol. and Ophthalmol., Univ. Miami School of Med., Miami, Fla.). Arch. Ophthalmol. 97:462, 1979.

Nystagmus intensities at various gaze angles were studied both preoperatively and postoperatively, using accurate ocular motility recordings, in three cases of congenital nystagmus. In addition to shifting the nystagmus null, the surgery broadened the null region and resulted in an overall reduction in nystagmus intensity at all gaze angles. Surgical rotation also resulted in improved visual acuity in all cases. The postoperative acuity at 0° was better than the preoperative acuity at both 0° and the patient's preferred gaze angle (i.e., the preoperative null angle). This was true not only for the two patients who showed an improved preoperative acuity with their head turn but also for the patient whose preoperative acuity did not substantially improve with her preferred head turn. Eye movement recordings have made it possible to accurately determine the amount of surgery required and to predict acuity increases even when undetectable during the preoperative clinical examination. (7 figures, 1 table, 27 references)—Authors' abstract

EVALUATION OF SOFT CONTACT LENS DISINFECTION IN THE HOME ENVIRONMENT. Pitts, R. E., and Krachmer, J. H.

(Dept. Ophthalmol., Iowa Lions Cornea Ctr., Univ. Hosp., Iowa City, Iowa). Arch. Ophthalmol. 97:470, 1979.

Recent reports of bacterial corneal ulcers in cosmetic soft contact lens wearers prompted an evaluation of one possible source of contamination, inadequate home disinfection of the lenses. Twenty-nine patients were asked to come for an early morning examination. They were told not to wear their lenses so their usual home disinfection procedure could be observed. On arrival, extensive bacterial and fungal cultures were taken of the solution in the previously unopened cases. Ten of the 29 patients (34.5%) had contaminated cases. Some patients did not follow the manufacturer's disinfection guidelines. The current disinfection procedure has repeatedly proven adequate in the laboratory. However, the practical fact is that a substantial percentage (34.5% in this study) of soft lens wearers are inadequately disinfecting their lenses. This serves as a possible source of contamination leading to bacterial corneal ulcers. (2 figures, 1 table, 10 references)—Authors' abstract

Australian Journal of Ophthalmology

THE TREATMENT OF BETA IRRADIATION NECROSIS OF THE SCLERA. Cameron, M. E. (79 Wickham Terrace, Brisbane, Australia). Aust. J. Ophthalmol. 6:86, 1978.

Late scleral necrosis after beta irradiation following pterygium excision is avoidable if cauterization is minimal, the area irradiated is no more than 12 x 6 mm and the dose does not exceed 1800 rad. Patching by conjunctival flaps of lamella corneal grafts is effective. Subconjunctival heparin increases vascularization. (1 figure, 1 reference)—Ronald Lowe

British Journal of Ophthalmology

RAPID SEROLOGICAL TEST FOR DIAGNOSIS OF CHLAMYDIAL EYE INFECTIONS. Darougar, S., Treharne, J.D., Minassian, D., El-Sheikh, H., Dines, R. J., and Jones, B. R. (Virus Lab. and WHO Ctr. for Reference and Res. on Trachoma and other Chlamydial Infections, Dept. Clin. Ophthalmol., Inst. Ophthalmol., and the External Diseases Clin., Moorfields Eye Hosp., London). Br. J. Ophthalmol. 62:503, 1978.

Since the incidence of genital infections by *Chlamydia trachomatis* is apparently on the increase in industrially developed countries, a higher incidence of ocular infections in their different clinical forms (Tric agent ophthalmia neonatorum, inclusion conjunctivitis, Tric agent punctate superficial keratitis or trachoma of genital transmission) is to be expected. This clinical polymorphism of the ocular manifestations - and to some extent also of the genital manifestations - creates a need for a simple, rapid, diagnostic test. Culture of the agent on irradiated McCoy cells is a rapid and sensitive method for the isolation of *chlmydia*, but requires an established laboratory and an experienced staff. In contrast, the micro-immunofluorescence test of Wang and Grayston has been modified so that it only requires 0.1 ml of blood or 0.05 ml of tears, taken up by cellulose sponges and kept at $^{+}4$° C until it is transported to the laboratory. The preparation of the antigen (inoculation of fertile hens' eggs with *chlamydia* and harvesting of the infected yolk-sac) is a simple procedure. Fluorescein-labelled antihuman IgA, IgG and IgM conjugates are already commercially available. Results of the method in 282 patients with various ocular infections are reported. The antibody titers in the serum of active, sexually transmitted ocular infections by *chlamydia* were significantly higher than in cases of other active, viral or bacterial, eye infections. The presence of antibodies IgG or IgA in the tears was clearly associated with paratrachoma. (2 figures, 2 tables, 19 references)—Peter C. Kronfeld

BILATERAL ACUTE RETINAL NECROSIS. Young, N. J. A., and Bird, A.C. (Moorfields Eye Hosp. and Inst. Ophthalmol., London). Br. J. Ophthalmol. 62:581, 1978.

The most striking feature in the retinas of the four patients described was the development of edematous, opaque, yellowish-white patches, first in one and a few days later in the second eye. In three of the cases the retinal lesions were, in the beginning, associated with a mild, transient, anterior uveitis. During the next three weeks the retinal lesions progressed, expanded and coalesced up to a sharp posterior border. Fluorescein angiography, when feasible, showed occlusion of some major vessels and capillary nonperfusion in the affected (i.e., the edematous) areas; dye leakage was observed from the few surviving major vessels crossing nonperfused retina and at the terminations of the occluded vessels as they entered the swollen tissue. Toward the end of the first month the retinal process appeared to regress in that the intense opacity cleared in spots, bringing mild changes of the pigment epithelium into view. As the clearing progressed, general narrowing and sheathing of the surviving vessels came to the fore. Concurrently with the clearing of the retina more and more debris appeared in the vitreous which the authors interpret as sloughing of necrotic retinal elements into the vitreous. In five of eight eyes the retina then became detached, with huge or multiple breaks at the posterior border of the original opacifications; the response to surgery was unfavorable. An extensive search for clues to the etiology of the disease was of no avail. The clinical picture is almost indistinguishable in the two cases of necrotizing vaso-occlusive retinitis reported by Willerson, in 1977. (5 figures, 16 references)—Peter C.Kronfeld

MACULAR ABNORMALITIES IN THE REATTACHED RETINA. Cleary, P. E., and Leaver, P. K. (Moorfields Eye Hosp., and Inst. of Ophthalmol., London). Br. J. Ophthalmol. 62:595, 1978.

In 66 patients whose retinal detachment had included the macula and responded favorably to surgery, the follow-up concentrated on the function and clinical appearance of the reattached central area. The visual acuity one year after surgery was found improved in 54 patients, the same as preoperatively in seven and worse in five patients. The appearance of the maculas was studied ophthalmoscopically, biomicroscopically, photographically and angiographically. The following categories could be recognized: normal appearance, cellophane maculopathy, minimal disturbance of the pigment epithelium (PE), cystoid change, macular hole, macular pucker, gross disturbance of the PE and cystoid macular edema. The term cystoid change refers to intraretinal cystoid spaces without angiographic abnormalities; in the majority of the cases so labelled the cystoid spaces were visible early and became overshadowed later by gross disturbances of the PE. Cellophane maculopathy is regarded as a mild degree of retinal pucker, with preretinal fibrosis as the common cause. True cystoid edema of the macula with leakage of fluorescein was observed early in 17 eyes, but subsequently resolved, with improvement of vision, in ten of the eyes. In a small number of eyes no detectable abnormality in the clinical appearance of the macula could account for the persistently reduced vision; defective receptor regeneration is considered a possible cause of the discrepancy. The general rule that duration of the detachment of more than eight weeks precludes a substantial return of vision was confirmed. (10 figures, 32 references)— Peter C. Kronfeld

CLEARANCE OF DEBRIS FROM THE IRIS THROUGH THE DRAINAGE ANGLE OF THE RABBIT'S EYE. Grierson, J., and Chisholm, I. A. (Inst. Ophthalmol., London, and Dept. Ophthalmol., Univ. of Saskatchewan, Canada). Br. J. Ophthalmol. 62:694, 1978.

Iris debris was produced experimentally by placing 12 to 15 burns with the Zeiss-Oberkochen photocoagulator in the right iris of 24 young rabbits. The clinical appearance of the lesions was at first that of discrete brown discolorations of the iris surface which in a short time gained in size and began to discharge blood, pigment and other debris into the anterior chamber. After ten days one or two burns had become through-and through holes. After 90 days the irides showed diffuse atrophy. At intervals varying from two hours to 90 days the eyes were enucleated and appropriate portions of the anterior segments were prepared for electron microscopy. During the first week after the photocoagulation two principal processes were observed in the trabecular meshwork (TM): 1) phagocytosis of particulate matter by native cells of the TM which thereby became active macrophages and wandered along with the normal current through giant vacuoles or nonvacuolar channels in the endothelium of Schlemm's canal, to be seen last in the lumina of the angular aqueous plexus which corresponds to Schlemm's canal in the primate; 2) the same passage of particulate material of the size of polymorphs or erythrocytes by the same routes could be observed without participation of macrophages. These phenomena are interpreted as a physiological safety mechanism which prevents rises in intraocular pressure as well as damage to the endothelium of Schlemm's canal. During the second week after the photocoagulation large, pigmented iris cells began to enter the TM, but were unable, because of their size, to pass through the physiological channels. This led to build-up of the large cells in the TM and to disruptions of its sheets which probably is the basis of the glaucoma associated with pigment dispersion syndromes or ocular contusions. (9 figures, 3 tables, 22 references)—Peter C. Kronfeld

Journal of Neurology, Neurosurgery and Psychiatry

BLUE-DOMED CYST WITH OPTIC NERVE COMPRESSION. Burnbaum, M. D., Harrison, J. W., Selhorst, J. B., and Young, H. F. (Dept. Neurol., Med. College of Virginia, Richmond, Va.). J. Neurol. Neurosurg. Psychiatry 41:987, 1978.

A previously healthy 43-year-old man awoke with severe right frontal headache. This was accompanied by pulsatile bursts of light in the right eye. Some five months later his vision was slightly reduced and there was an inferior altitudinal defect. A complete diagnostic workup was done including right carotid arteriogram and all was normal. When the headaches recurred corticosteroids were prescribed with success. After about a year the prednisone was discontinued and severe right fronto-orbital pain occurred accompanied by an abrupt loss of vision. At this time a CT scan showed a small midline suprasellar density. Surgical exploration revealed a small dark blue cyst underlying the right optic nerve. These cysts because of their small size are extremely difficult to diagnose as this case illustrates. Computerized tomography may be helpful but the diagnostic procedure of choice is pneumoencephalography with AP polytomography. (5 figures, 1 table, 7 references)—David Shoch

Journal of Ultrastructure Research

MOLECULAR STRUCTURE OF OUTER SEGMENT DISKS IN PHOTORECEPTOR CELLS. Sjöstrand, F. S., and Kreman, M. (Molecular Biol. Inst. and Dept. Biol., Univ. California, Los Angeles, Calif.). J. Ultrastruct. Res. 65:195, 1978.

The outer segment disk membranes consist of two layers with greatly different physical properties and with each layer measuring about 50 Å in thickness. Each disk membrane consists of one lipid bilayer and one monolayer of photopigment molecules dispersed in an aqueous medium of small volume. A particulate structure at the edge of the disk makes this part of the disk appear structurally different from the disk membrane. The outer segment disk membrane differs structurally extensively from the inner mitochondrial membranes, and the differences are interpreted to reflect the very different functions at a molecular level of these two types of membranes. (14 figures, 76 references)—Authors' abstract

Klinische Monatsblaetter für Augenheilkunde

HERPETIFORM BILATERAL EPITHELIAL CORNEAL DYSTROPHY CAUSED BY TYROSINEMIA (RICHNER-HANHART SYNDROME). (German) Jaeger, W., Gallasch, G., Schnyder, U. W., Lutz, P., and Schmidt, H. (Univ. Eye Clin., Dermatology Clin., and Pediatr. Clin., Heidelberg, West Germany). Klin. Monatsbl. Augenheilkd. 173:506, 1978.

Tyrosinemia, an inherited disorder of metabolism, is associated with a definite clinical syndrome involving multiple organs. The eye findings appear first and allow a diagnosis to be made at an early age when dietary treatment is still possible. There is a dendritic epithelial corneal dystrophy which appears in the first year of life. This resembles a dendrite caused by herpes simplex, but can be distinguished because it is bilateral, corneal sensitivity is normal, blepharospasm and photophobia may be extreme, and the figure remains without change for a long time.

Other signs, appearing later in the school age child, are hyperkeratosis of palms and feet, and mental retardation. The diagnosis in confirmed by finding serum tyrosine greater than 10 mg/100 ml. The exact enzyme defect is unknown. Dietary restriction in a child leads to reversal of the corneal abnormality; however, if the diagnosis is made in adult patients, dietary therapy is of limited value. (4 figures, 32 references)—Peter R. Egbert

MEASURING THE BREADTH OF FUSION IN REDUCED MONOCULAR AND BINOCULAR VISUAL ACUITY. (German) Aust, W. (Teaching Hosp. Eye Clin., Kassel, West Germany). Klin. Monatsbl. Augenheilkd. 173:671, 1978.

The range of fusion was measured with the synoptophore with different objects in 100 students. The visual acuity of the tested persons was reduced monocularly and binocularly by fogging occluders. The range of fusion decreases more strongly in the case of monocularly reduced vision than in the case of binocularly reduced vision to the same degree. Therefore it is not the absolute value of the reduced vision which is responsible for the decrease in the range of fusion but rather the difference between the visual acuity of both eyes. This result is important for the therapy with fogging occluders, prism foils and penalisation methods. (2 figures, 2 tables, 2 references)—Author's abstract

PRIMARY RECONSTRUCTION OF INJURED CANALICULI. (German) Hanselmayer, H. (Univ. Eye Clin., Graz, Austria). Klin. Monatsbl. Augenheilkd. 173:712, 1978.

Follow up of 125 patients with injuries of the canaliculi which were treated by different methods from 1960–1970 showed that 82.4% of the injured canaliculi were patent. Recently, by using microsurgical adaptation of the ends of the canaliculus, more than 90% patency has been achieved. The author's preferred method is to pass a silicone tube over a silk suture so that a circle is formed through both canaliculi and the lacrimal sac. He next sutures the canaliculus ends directly with at least three sutures and closes the subcutaneous tissue and skin. The silicone stent is left in place three to ten weeks.

A delay of surgery for up to several days did not decrease the success rate, so that although immediate repair is recommended, delayed repair is also possible. (2 figures, 1 table, 6 references)—Peter Egbert

OPERATIONS AND OCCLUSION TREATMENT IN UNILATERAL CONGENITAL CATARACT. (German) Meyner, E. M. (Univ. Eye Clin. of Tubingen, Tubingen, West Germany). Klin. Monatsbl. Augenheilkd. 173:737, 1978.

Since 1967, 97 children with unilateral cataracts were followed prospectively to evaluate the functional results of surgery. Microphthalmos or other associated severe defects were present in 37 of the eyes. Cataract surgery was performed on 54 children; 22 of the operations were in the first or second year of life. Operative and postoperative complications were rare. Strict occlusion of the good eye was always attempted. In 31, or nearly half of the children, the occlusion was not conscientiously carried out by the parents; in 15 others, the occlusion eventually had to be stopped because the child remained too sad or angry over an extended period; the remaining 18 children tolerated the occlusion well after a few days. Of these 18 who had strict occlusion for a long time, ten had no improvement and poor vision, six improved to useful vision but with eccentric fixation and only two achieved good vision with central fixation.

Although reading vision or binocular fusion is seldom achieved in these patients, occlusion therapy can occasionally achieve useful vision. It is apparent in a few days from the start of occlusion whether the treatment will be useful or not. (2 tables, 12 references)—Peter Egbert

Medical Journal of Australia

OCULAR MANIFESTATIONS AND COMPLICATIONS OF ACUTE METHYL ALCOHOL

INTOXICATION. Dethlefs, R., and Naraqi, S. (Port Moresby Gen. Hosp. and Univ. Papua, New Guinea). Med. J. Aust. 2:483, 1978.

Acute methyl alcohol intoxication occurred in a group of 24 men. About a third of the group had no ocular complications, a third had transient abnormalities and the remaining third had permanent ocular damage. In general the incidence and severity of the ocular findings vary directly with the presence of acidosis and with the reported volume of methanol ingested. It is probable that the level of acidosis simply reflects the level of either formaldehyde or formate in the blood which are probably the immediate causes of the ocular toxicity. (3 tables, 9 references)—David Shoch

Ophthalmic Surgery

THERMAL SCLEROSTOMY AND POSTERIOR LIP SCLERECTOMY: A COMPARATIVE STUDY. Marion, J. R., and Shields, M. B. (Duke Univ. Eye Ctr., Durham, N. C.). Ophthalmic Surg. 9:67, 1978.

Eighty-two consecutive filtering procedures (47 thermal sclerostomies and 35 posterior lip sclerectomies), performed by senior staff and residents at the Duke University Eye Center between 1972–1976, were reviewed and compared. Operative and early postoperative complications were more common with the posterior lip sclerectomies. However, late postoperative complications were equal for the two groups, with cataracts occurring in approximately 30% of both categories.

A final intraocular pressure of 20 mm Hg or less, with or without medication, was achieved in 66% of the thermal sclerostomies and 85.7% of the posterior lip sclerectomies. Residents did not perform as well as the senior staff when using thermal sclerostomy (60% vs. 70%, respectively), but performed equally well when using posterior lip sclerectomy (85.7% for both groups). In comparing black and white patients, the final intraocular pressures were the same in both groups. (2 figures, 15 tables, 6 references)—Authors' abstract

Ophthalmology

IRIDOCYCLECTOMY FOR MELANOMAS OF THE CILIARY BODY: A FOLLOW-UP STUDY OF PATHOLOGY AND SURGICAL MORBIDITY. Forrest, A. W., Keyser, R. B., and Spencer, W. H. (Edward S. Harkness Eye Inst. and Columbia Presbyterian Med. Ctr., New York, N. Y.). Ophthalmol. 85:1237, 1978.

One hundred seven cases of ciliary body melanoma treated by iridocyclectomy were studied with respect to the effects of surgical treatment upon the eye and the subsequent tumor behavior; 6% were enucleated because of surgical complications. Forty-two percent had final postoperative visual acuity of 20/40 or better and 95 of the melanomas were spindle cell A or B. Three tumor deaths occurred within four years, one caused by large spindle cell B tumor and the other two resulted from epithelioid tumors. In one-third of the cases complete excision was noted, however, even in the remaining two-thirds only 20% showed a regrowth of the tumor during the follow-up period of 14 years. Recurrence developed within four years in all but two cases. The data suggest that the cell type of the tumor is the primary prognostic feature, and surgical intervention via iridocyclectomy does not increase the incidence of metastases. No cases of local orbital recurrence developed. (8 figures, 4 tables, 3 references)—Authors' abstract

Plastic and Reconstructive Surgery

REVIEW OF 1,000 MAJOR FACIAL FRACTURES AND ASSOCIATED INJURIES. Luce, E. A., Tubb, T. D., and Moore, A. M. (Dept. Surg.—Plast., Univ. of Kentucky Med. Ctr., Lexington, Ky.). Plast. Reconstr. Surg. 63:26, 1979.

The authors studied the incidence of major (life-threatening) and minor injuries associated with facial fractures. The circumstances of the injury were divided into a high velocity group, e.g., motor vehicle accidents, and a low velocity group, e.g., assault. The high velocity group was further subdivided into groups of patients who sustained fractures of facial bones having either a high or low resistance to impact. Of the facial bones, the supraorbital ridges have the greatest resistance to impact. Patients with fractures of the supraorbital ridge had a 70 per cent incidence of associated major injury, such as pulmonary, central nervous system or intra-abdominal complications. The overall incidence of ocular injuries was 2.7 per cent. (2 figures, 1 table, 16 references)—Gerald J. Harris

THE CROCODILE TEAR SYNDROME. McCoy, F. J., and Goodman, R. C. (Dept. Surg.—Plast., Univ. Missouri School of Med., Kansas City, Mo.). Plast. Reconstr. Surg. 63:58, 1979.

The syndrome of unilateral lacrimation associated with eating or drinking most commonly follows a traumatic facial paralysis or Bell's palsy. The mechanism probably involves misdirection of regenerating nerve fibers so that fibers originally destined for the salivary glands find their way to the lacrimal gland. The authors describe two cases which they treated by partial resection of the palpebral lobe of the lacrimal gland. (3 figures, 16 references)—Gerald J. Harris

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NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

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For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least three months before the date of occurrence.

UNITED STATES—UNION OF SOVIET SOCIALIST REPUBLICS: FORMAL COLLABORATION IN RESEARCH OF EYE DISEASES

Joseph A. Califano, Jr., Secretary of Health, Education and Welfare announced on March 7 that the United States and Russia have begun formal collaboration in research on eye diseases. Coordinating the cooperative vision research program for the United States is Carl Kupfer, M.D., Director of the National Eye Institute. Professor Michail M. Krasnov, M.D., Director of the All-Union Scientific Research Institute of Eye Diseases of the Soviet Ministry of health, is coordinator for the U.S.S.R.

CONGRESS OF THE INTERNATIONAL SOCIETY OF GEOGRAPHICAL OPHTHALMOLOGY

The Congress of the International Society of Geographical Ophthalmology will be held in Rio De Janeiro, Brazil, Sept. 9–14, 1979. For further information, write James P. Ganley, M.D., University of Arizona Health Sciences Center, Tucson, AZ 85724.

SECOND CONGRESS OF THE INTERNATIONAL SOCIETY OF PEDIATRIC OPHTHALMOLOGY

The Second Congress of the International Society of Pediatric Ophthalmology will be held in Parma, Italy, June 20–June 24, 1979. For further information, write Heskel M. Haddad, M.D., Secretary, 1200 Fifth Avenue, New York, NY 10029.

NEW YORK MEDICAL COLLEGE: SEMI-ANNUAL VII NERVE SURGICAL DISSECTION COURSE

The New York Medical College will sponsor the Semi-Annual VII Nerve Surgical Dissection Course in Elmsford, New York Nov. 18–20, 1979. The fee is $500. For further information, write Ms. P. Tamkin, c/o P. Guibor, M.D., 630 Park Ave., New York, NY 10021; telephone (212) 734-1010.

MOORFIELDS EYE HOSPITAL DEPARTMENT OF CLINICAL OPHTHALMOLOGY—INSTITUTE OF OPHTHALMOLOGY: COURSE IN OPHTHALMIC MICROSURGERY

The Department of Clinical Ophthalmology, Institute of Ophthalmology, will conduct a course in ophthalmic microsurgery Oct. 24-26, 1979 in London England. Fee for the course is $160. The closing date for applications is Sept. 21, 1979. For further information, write Mrs. P. M. Usher, Microsurgical Course Secretary, Department of Clinical Ophthalmology, Moorfields Eye Hospital, City Road, London EC1V 2PD, England.

WORLD CONVENTION OF MICROSURGERY

The World Convention on Microsurgery will be held in Singapore, Dec. 2-5, 1979. For further information, write Arthur S. M. Lim, M.D., Chairman, World Convention on Microsurgery, Mount Elizabeth Hospital, Mount Elizabeth, Singapore 10.

XI CONGRESO ARGENTINO DE OFTALMOLOGIA

The XI Congreso Argentino de Oftalmologia will be held Nov. 2-8, 1979, in Mar del Plata, Argentina. For further information, write Hugo D. Nano, M.D., Casilla de Correo 736-7600 Mar Del Plata, PCIA. BS. AS. Argentina.

PARK RIDGE HOSPITAL, ROCHESTER, NEW YORK: FOURTH ANNUAL OPHTHALMIC SEMINAR

The Department of Ophthalmology at Park Ridge Hospital, Rochester, New York, will sponsor the Fourth Annual Ophthalmic Seminar Aug. 10 and 11, 1979. For further information, write Ms. Katherine Sgabelloni, Coordinator, Ophthalmic Services, Park Ridge Hospital, 1555 Long Pond Road, Rochester, NY 14626.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY: ANNUAL MEETING

The Montana Academy of Oto-Ophthalmology will hold its annual meeting Aug. 3 and 4, 1979, in Kalispell, Montana. The program topic will be External Disease. For further information, write Steve W. Weber, M.D., P. O. Box 1080, Kalispell, MT 59901.

NEW YORK MEDICAL COLLEGE: SEMI-ANNUAL OCULOPLASTIC SURGICAL DISSECTION COURSE

The New York Medical College will sponsor the Semi-Annual Oculoplastic Surgical Dissection course Nov. 14-17, 1979. The fee is $600. For further information, write Ms. P. Tamkin, c/o P. Guibor, M.D., 630 Park Ave., New York, NY 10021 telephone: (212) 734-1010.

FIFTH ANNUAL COLBY COLLEGE OPHTHALMOLOGY SEMINAR

Colby College, in conjunction with Mid-Maine Medical Center, Waterville, Maine, will present a four-day post-graduate seminar on glaucoma Aug. 5-9, 1979. For further information, write Robert H. Kany, Director, Special Programs, Colby College, Waterville, ME 04901.

UNIVERSITY OF PUERTO RICO MERCY HOSPITAL: APHAKIA SYMPOSIUM

The University of Puerto Rico Mercy Hospital will sponsor an Aphakia Symposium Dec. 26, 1979, to Jan. 1, 1980. The three main topics are: Aphakic glasses, extended wear contact lenses, and intraocular lenses. Free papers related to the three topics are invited. For further information, write Mr. Stuart Hama, 333 St. Paul's Place, Baltimore, MD 21202.

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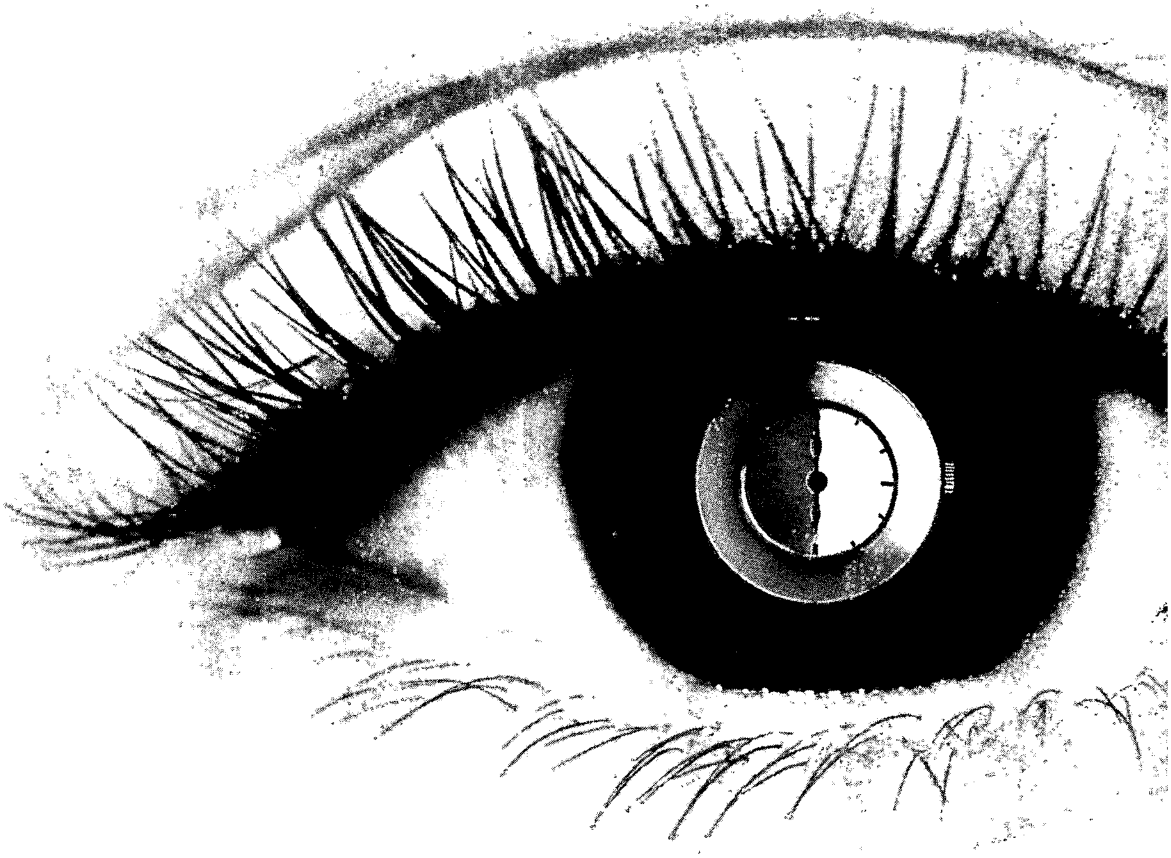

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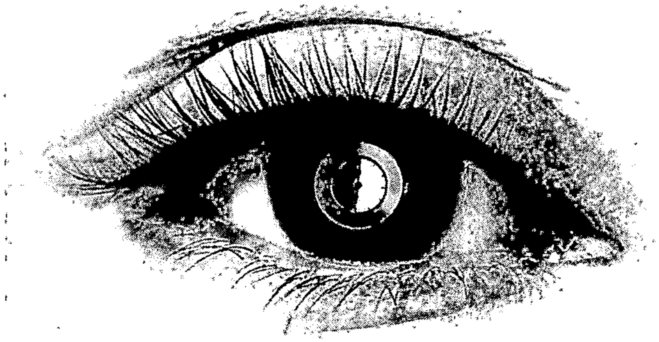

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2. Succinylcholine should be administered only with great caution, if at all, prior to or during general anesthesia to patients receiving anticholinesterase medication because of possible respiratory or cardiovascular collapse.

3. Caution should be observed in treating glaucoma with PHOSPHOLINE IODIDE in patients who are at the same time undergoing treatment with systemic anticholinesterase medications for myasthenia gravis, because of possible adverse additive effects.
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2. Where there is a quiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and ciliary muscle contraction that may occur.

3. While systemic effects are infrequent, proper use of the drug requires digital compression of the nasolacrimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation

4. Temporary discontinuance of medication is necessary if salivation, urinary incontinence, diarrhea, profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities occur.

5. Patients receiving PHOSPHOLINE IODIDE who are exposed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin. During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable.

6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer, pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.

7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.

8. PHOSPHOLINE IODIDE (echothiophate iodide) should be used with great caution, if at all, where there is a prior history of retinal detachment.
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2. Stinging, burning, lacrimation, lid muscle twitching, conjunctival and ciliary redness, browache, induced myopia with visual blurring may occur.

3. Activation of latent iritis or uveitis may occur.

4. Iris cysts may form, and if treatment is continued, may enlarge and obscure vision. This occurrence is more frequent in children. The cysts usually shrink upon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Rarely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative esotropia.

5. Prolonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.

6. Lens opacities occurring in patients under treatment for glaucoma with PHOSPHOLINE IODIDE have been reported and similar changes have been produced experimentally in normal monkeys. Routine examinations should accompany clinical use of the drug.

7. Paradoxical increase in intraocular pressure may follow anticholinesterase instillation. This may be alleviated by prescribing a sympathomimetic mydriatic such as phenylephrine.
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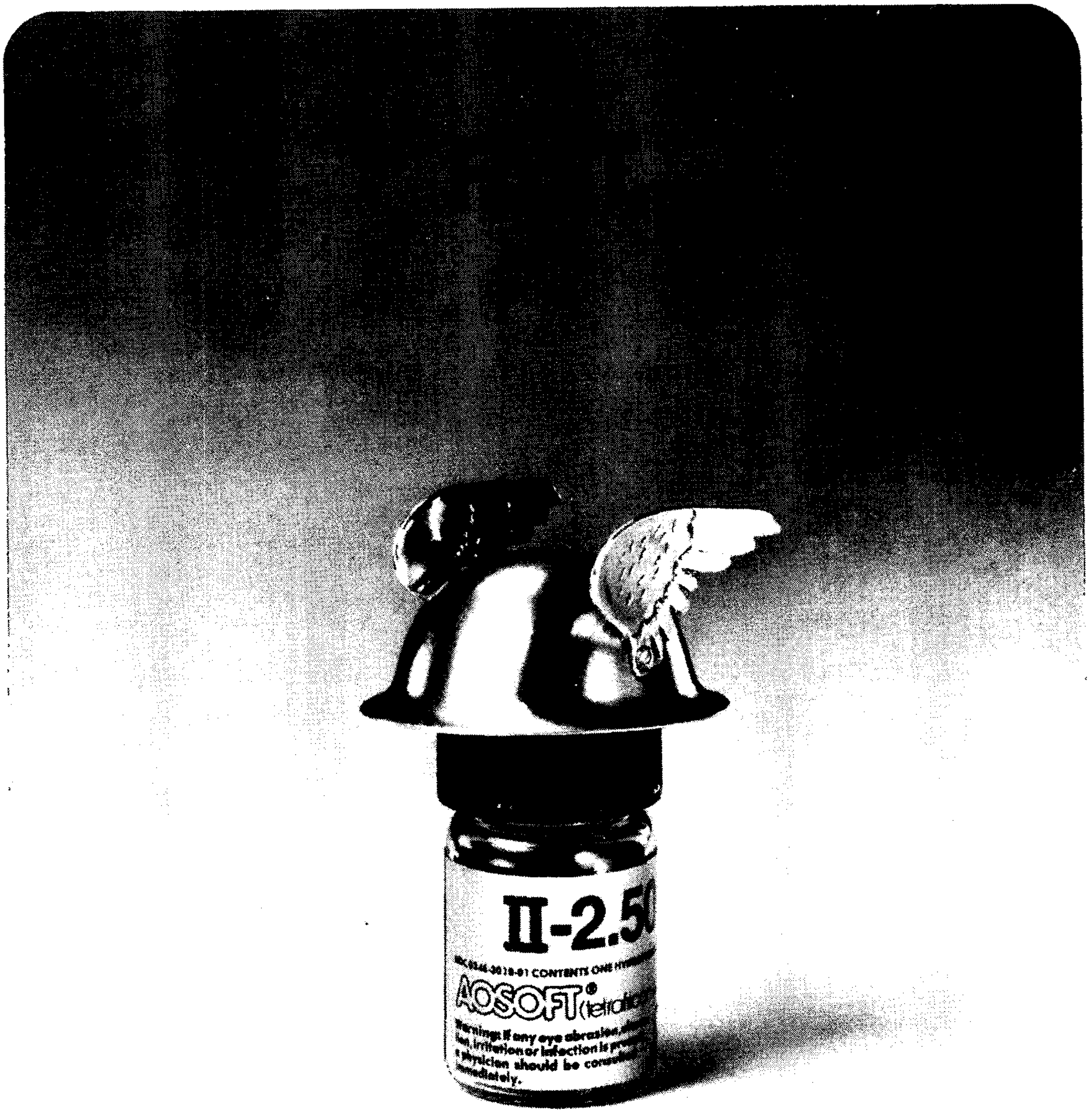

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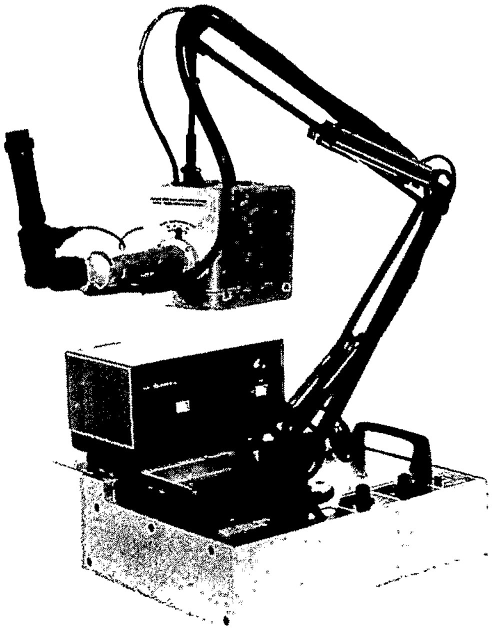

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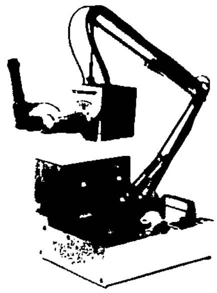

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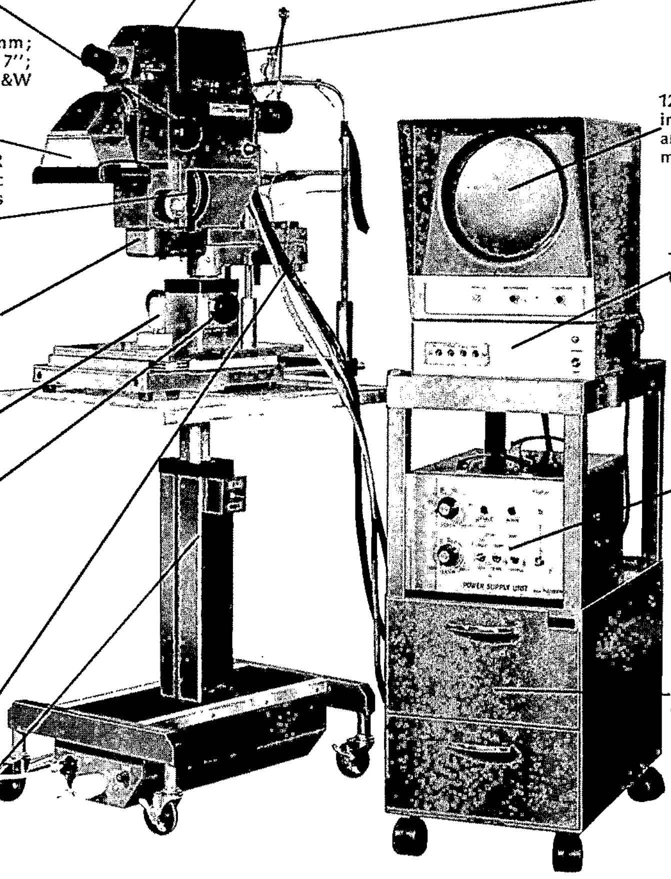

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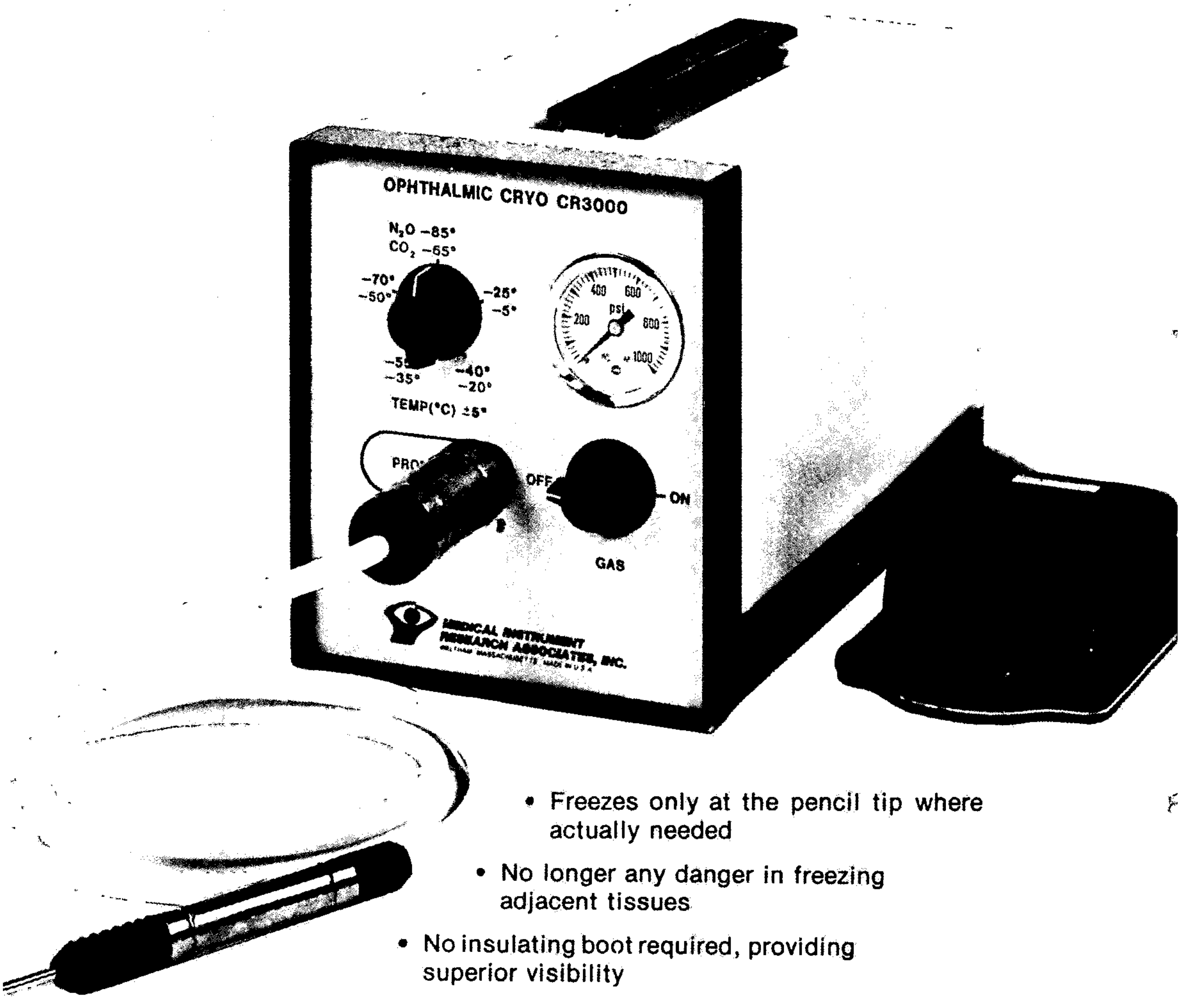

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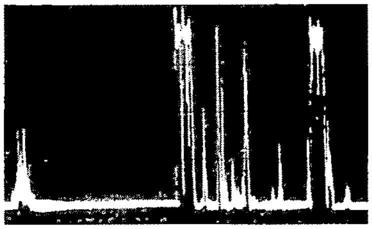

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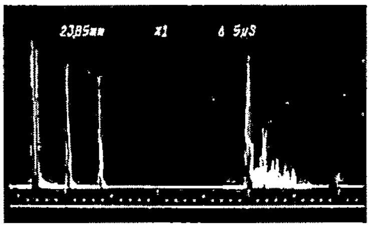

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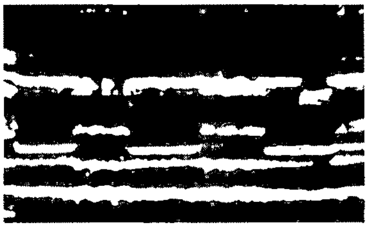

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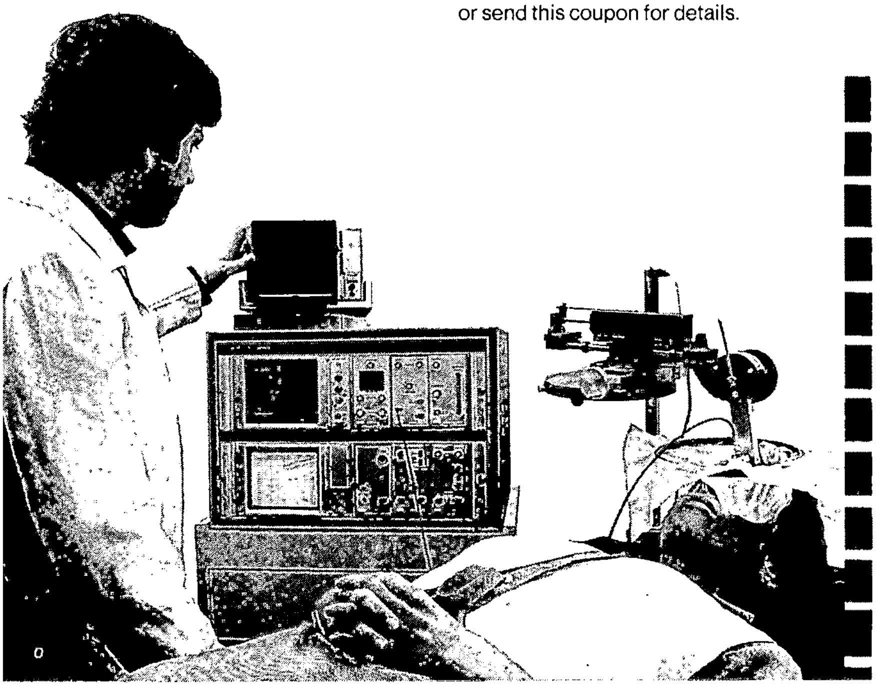

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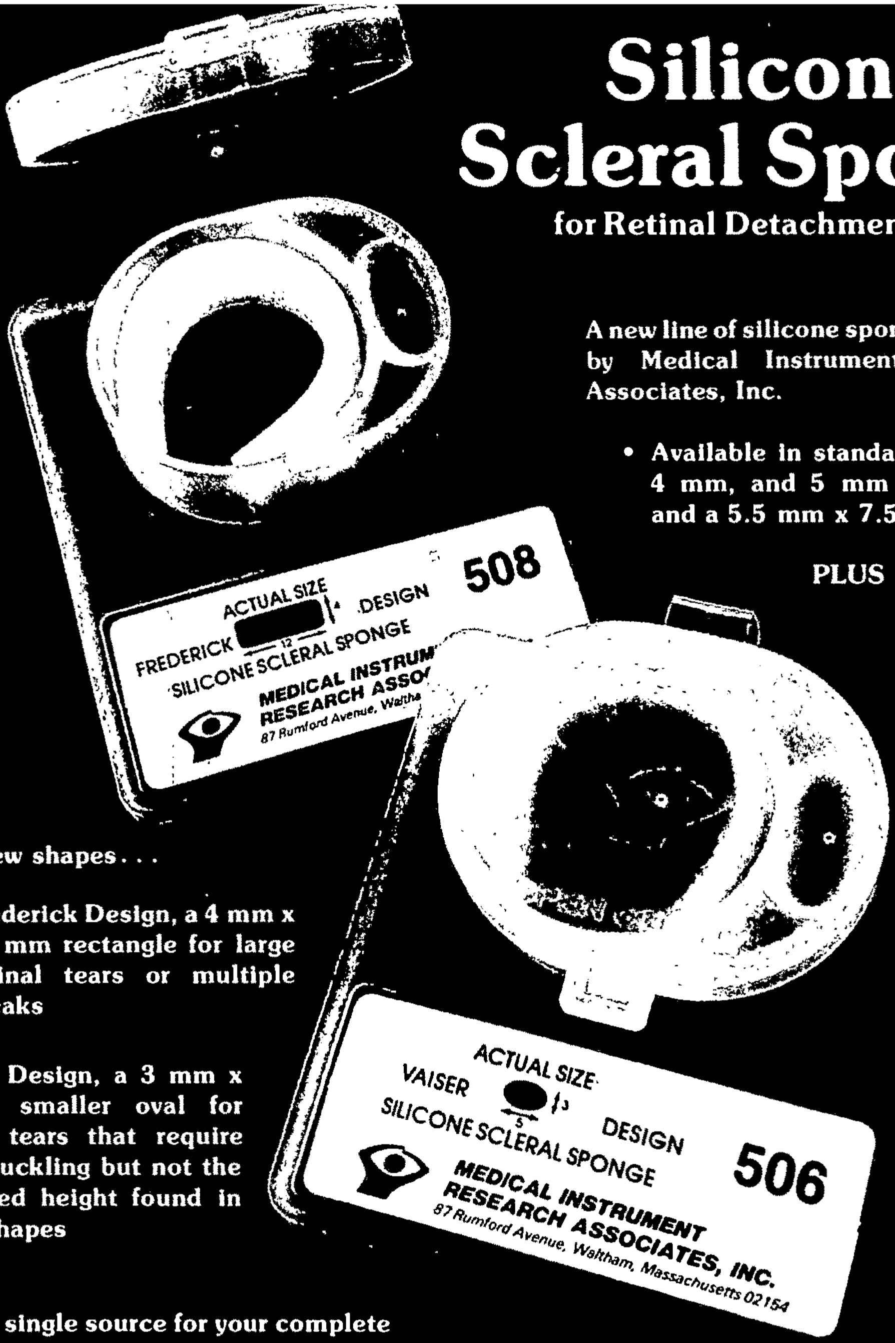

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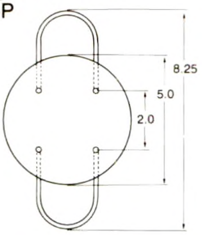

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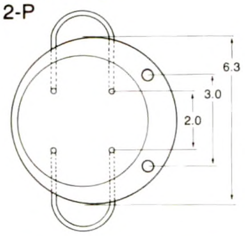

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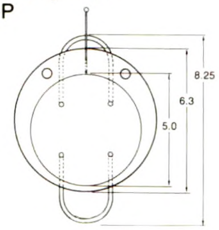

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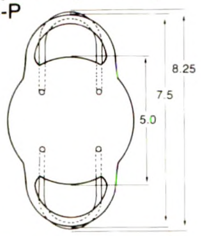

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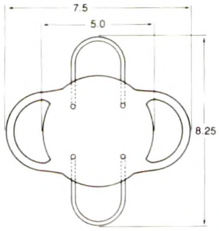

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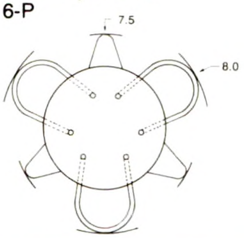

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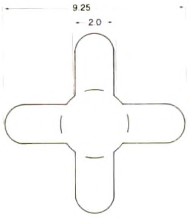

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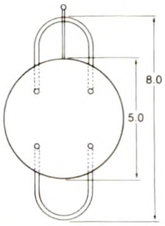

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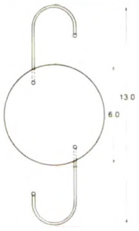

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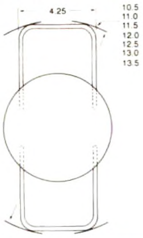

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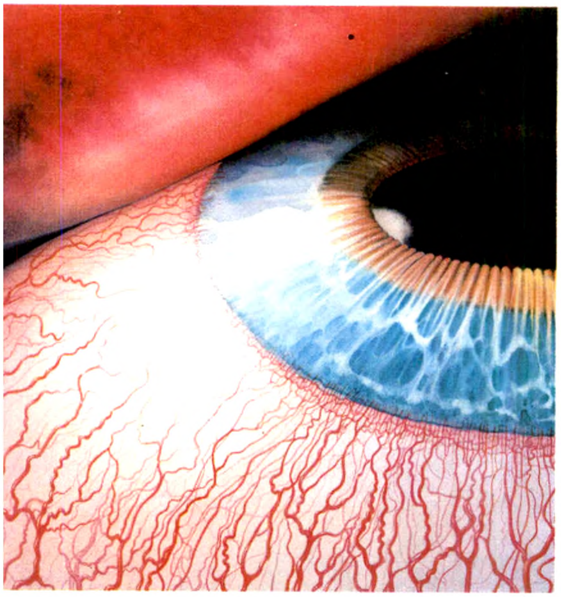

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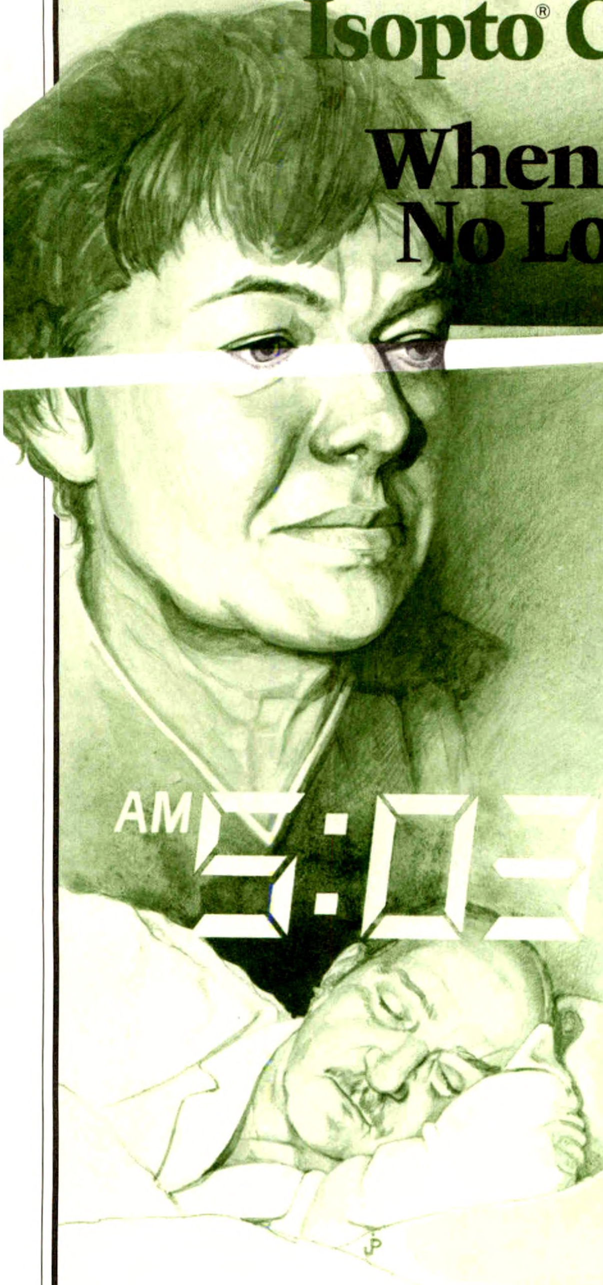

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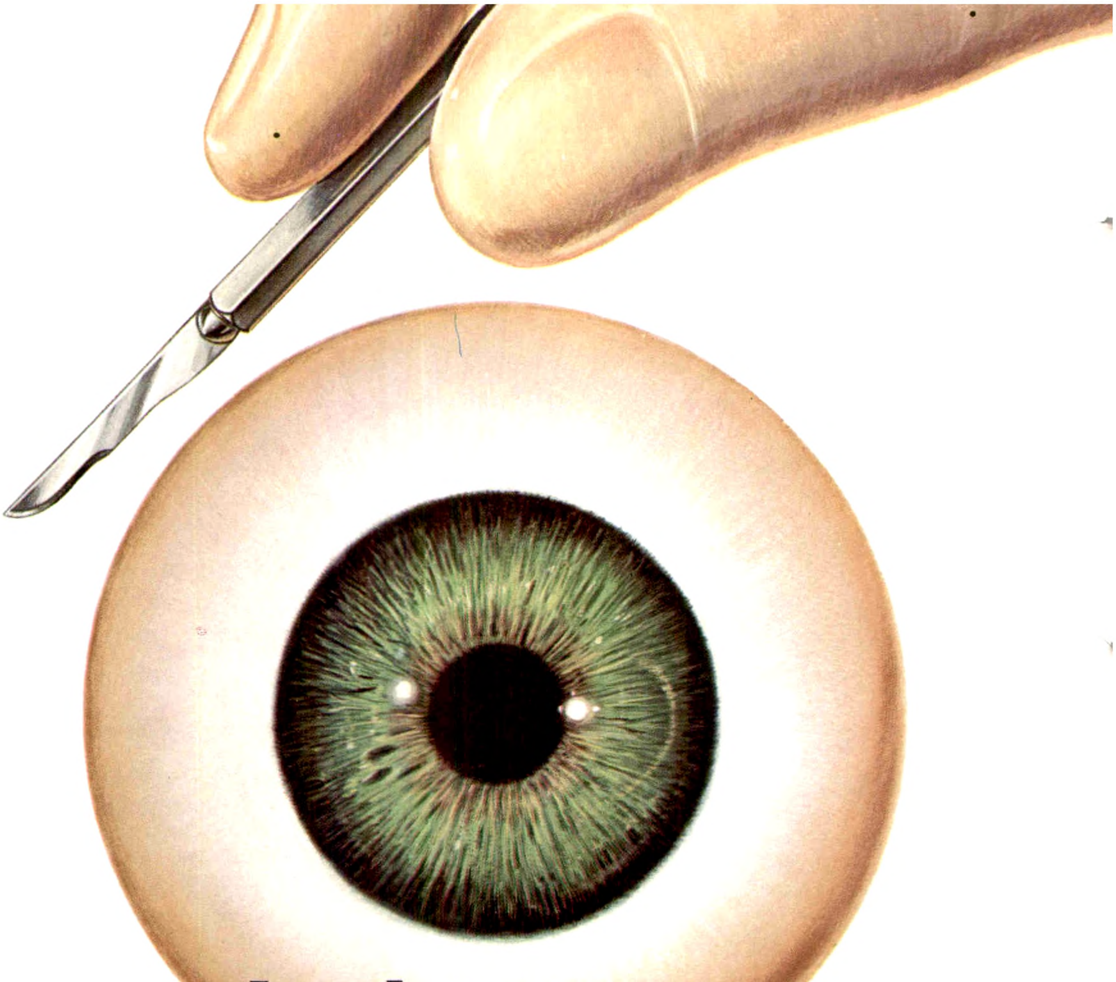

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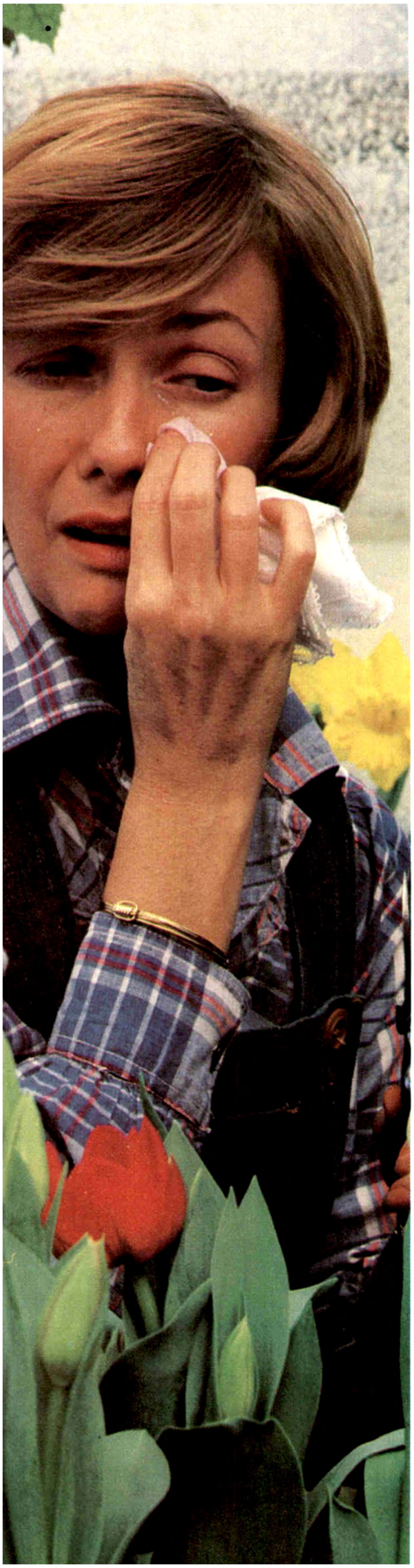

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"Possibly" effective: For relief of ocular irritation and/or congestion or for the treatment of allergic, inflammatory, or infectious ocular conditions. Final classification of the less-than-effective indication requires further investigation.

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VIRA-A® (vidarabine ophthalmic ointment), 3%

Description. Vira-A (vidarabine, adenine arabinoside, or Ara-A) is an antiviral drug for topical ophthalmic administration. The chemical name is 9-β-D-arabinofuranosyladenine. Each gram of the ophthalmic ointment contains 30 mg of vidarabine monohydrate equivalent to 28.11 mg of vidarabine in a sterile, inert, petrolatum base.

Clinical Pharmacology. Vira-A is a purine nucleoside obtained from fermentation cultures of *Streptomyces antibioticus*. Vira-A possesses *in vitro* and *in vivo* antiviral activity against Herpes simplex types 1 and 2, Varicella-Zoster, and Vaccinia viruses. Except for Rhabdovirus and Oncornavirus, Vira-A does not display *in vitro* antiviral activity against other RNA or DNA viruses, including Adenovirus.

The antiviral mechanism of action has not been established. Vira-A appears to interfere with the early steps of viral DNA synthesis. Vira-A is rapidly deaminated to arabinosylhypoxanthine (Ara-Hx), the principal metabolite. Ara-Hx also possesses *in vitro* antiviral activity but this activity is less than that of Vira-A. Because of the low solubility of Vira-A, trace amounts of both Vira-A and Ara-Hx can be detected in the aqueous humor only if there is an epithelial defect in the cornea. If the cornea is normal, only trace amounts of Ara-Hx can be recovered from the aqueous humor.

Systemic absorption of Vira-A should not be expected to occur following ocular administration and swallowing lacrimal secretions. In laboratory animals, Vira-A is rapidly deaminated in the gastrointestinal tract to Ara-Hx.

In contrast to topical idoxuridine, Vira-A demonstrated less cellular toxicity in the regenerating corneal epithelium of the rabbit.

Indications and Usage. Vira-A Ophthalmic Ointment, 3%, is indicated for the treatment of acute keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus types 1 and 2. Vira-A is also effective in superficial keratitis caused by Herpes simplex virus which has not responded to topical idoxuridine or when toxic or hypersensitivity reactions to idoxuridine have occurred. The effectiveness of Vira-A Ophthalmic Ointment, 3%, against stromal keratitis and uveitis due to Herpes simplex virus has not been established.

The clinical diagnosis of keratitis caused by Herpes simplex virus is usually established by the presence of typical dendritic or geographic lesions on slit-lamp examination. In controlled and uncontrolled clinical trials, an average of seven and nine days of continuous Vira-A Ophthalmic Ointment, 3%, therapy was required to achieve corneal re-epithelialization. In the controlled trials, 70 of 81 subjects (86%) re-epithelialized at the end of three weeks of therapy. In the uncontrolled trials, 101 of 142 subjects (71%) re-epithelialized at the end of three weeks. Seventy-five percent of the subjects in these uncontrolled trials had either not healed previously or had developed hypersensitivity to topical idoxuridine therapy.

The following topical antibiotics: gentamicin, erythromycin, chloramphenicol; or topical steroids: prednisolone or dexamethasone, have been administered concurrently with Vira-A Ophthalmic Ointment, 3%, without an increase in adverse reactions.

Normally, corticosteroids alone are contraindicated in Herpes simplex virus infections of the eye. If Vira-A Ophthalmic Ointment, 3%, is administered concurrently with topical corticosteroid therapy, corticosteroid-induced ocular side effects must be considered. These include corticosteroid-induced glaucoma or cataract formation and progression of a bacterial or viral infection.

Vira-A is not effective against RNA virus or adenoviral ocular infections. Vira-A is also not effective against bacterial, fungal, or chlamydial infections of the cornea or nonviral trophic ulcers.

Contraindication. Vira-A Ophthalmic Ointment, 3%, is contraindicated in patients who develop hypersensitivity reactions to it.

Warnings. ***Use in Pregnancy:*** Vira-A (vidarabine) parenterally is teratogenic in rats and rabbits. Ten percent Vira-A ointment applied to 10% of the body surface during organogenesis induced fetal abnormalities in rabbits. When 10% Vira-A ointment was applied to 2 to 3% of the body surface of rabbits, no fetal abnormalities were found. This dose greatly exceeds the total recommended ophthalmic dose in humans. The possibility of embryonic or fetal damage in pregnant women receiving Vira-A (vidarabine ophthalmic ointment), 3%, is remote. The topical ophthalmic dose is small, and the drug relatively insoluble. Its ocular penetration is very low. However, a safe dose for a human embryo or fetus has not been established. Consequently, Vira-A should be used only when clearly indicated.

It is not known whether Vira-A is excreted in human milk. As a general rule, because many drugs are excreted in human milk, nursing should not be undertaken while a patient is under treatment. However, excretion of vidarabine in breast milk is unlikely because Vira-A is rapidly deaminated in the gastrointestinal tract.

Mutagenic Potential: Results of *in vitro* experiments indicate that vidarabine can be incorporated into mammalian DNA and can induce mutation in mammalian cells (mouse L5178Y cell line). Thus far, *in vivo* studies have not been as conclusive, but there is some evidence (dominant lethal assay in mice) that vidarabine may be capable of producing mutagenic effects in male germ cells.

It has also been reported that vidarabine causes chromosome breaks and gaps when added to human leukocytes *in vitro*. While the significance of these effects in terms of mutagenicity is not fully understood, there is a well-known correlation between the ability of various agents to produce such effects and their ability to produce heritable genetic damage.

Oncogenic Potential: Chronic parenteral (IM) studies of vidarabine have been conducted in mice and rats.

In the mouse study, there was a statistically significant increase in liver tumor incidence among the vidarabine-treated females. In the same study, some vidarabine-treated male mice developed kidney neoplasia. No renal tumors were found in the vehicle-treated control mice or the vidarabine-treated female mice.

In the rat study, intestinal, testicular, and thyroid neoplasia occurred with greater frequency among the vidarabine-treated animals than in the vehicle-treated controls. The increases in thyroid adenoma incidence in the high-dose (50 mg/kg) males and the low-dose (30 mg/kg) females were statistically significant.

Hepatic megalocytosis, associated with vidarabine treatment, has been found in short- and long-term rodent (rat and mouse) studies. It is not clear whether or not this represents a preneoplastic change.

The recommended frequency and duration of administration should not be exceeded (see Dosage and Administration).

Precautions. The diagnosis of keratoconjunctivitis due to Herpes simplex virus should be established clinically prior to prescribing Vira-A Ophthalmic Ointment, 3%.

Patients should be forewarned that Vira-A Ophthalmic Ointment, 3%, like any ophthalmic ointment, may produce a temporary visual haze.

Although viral resistance to Vira-A has not been observed, this possibility may exist.

Adverse Reactions. Lacrimation, foreign-body sensation, conjunctival injection, burning, irritation, superficial punctate keratitis, pain, photophobia, punctal occlusion, and sensitivity have been reported with Vira-A Ophthalmic Ointment, 3%. The following have also been reported but appear disease-related: uveitis, stromal edema, secondary glaucoma, trophic defects, corneal vascularization, and hyphema.

Overdosage. Acute massive overdosage by oral ingestion of the ophthalmic ointment has not occurred. However, the rapid deamination to arabinosylhypoxanthine should preclude any difficulty. The oral LD_{50} for vidarabine is greater than 5,020 mg/kg in mice and rats. No untoward effects should result from ingestion of the entire contents of a tube.

Overdosage by ocular instillation is unlikely because any excess should be quickly expelled from the conjunctival sac. Too frequent administration should be avoided.

Dosage and Administration. Administer approximately one half inch of Vira-A Ophthalmic Ointment, 3%, into the lower conjunctival sac five times daily at three-hour intervals.

If there are no signs of improvement after 7 days, or complete re-epithelialization has not occurred by 21 days, other forms of therapy should be considered. Some severe cases may require longer treatment.

After re-epithelialization has occurred, treatment for an additional seven days at a reduced dosage (such as twice daily) is recommended in order to prevent recurrence.

How Supplied. N 0071-3677-07 (Stock 18-1677-139)

Vira-A Ophthalmic Ointment, 3%, is supplied sterile in ophthalmic ointment tubes of 3.5 g. The base is a 60:40 mixture of solid and liquid petrolatum. **UK**

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in patients resistant or
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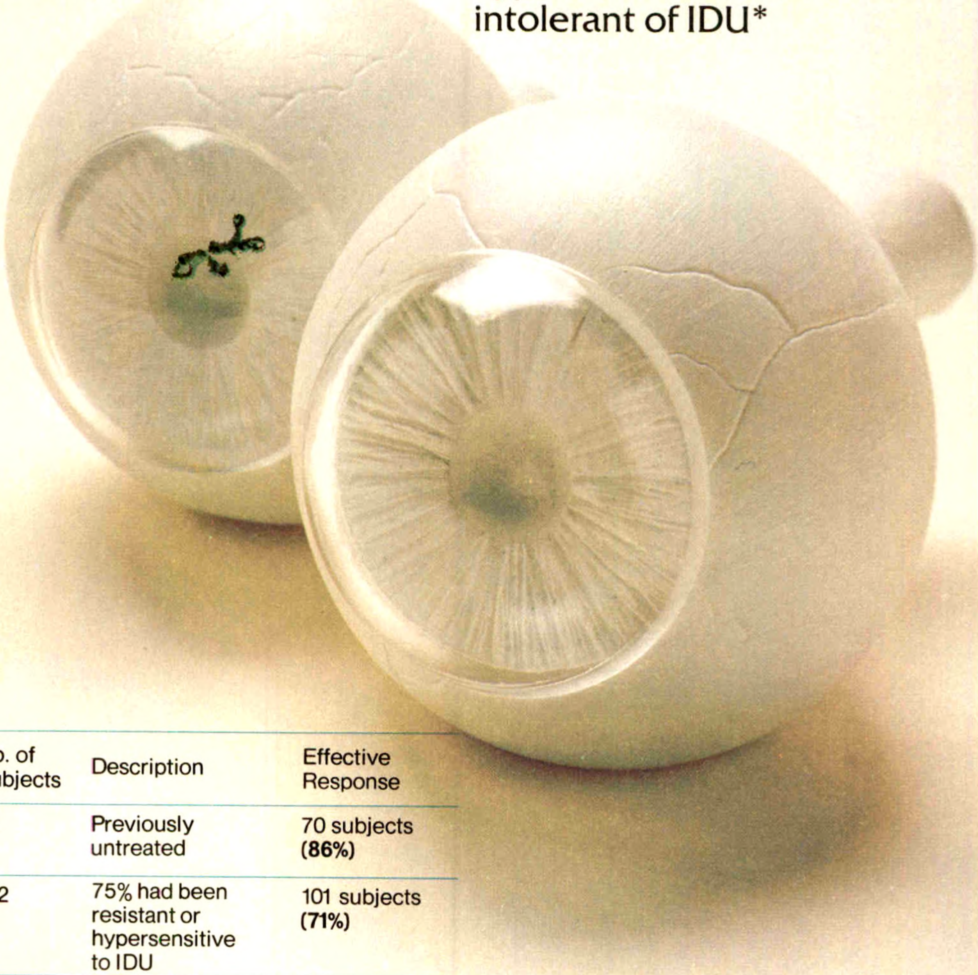

inical Evidence	No. of Subjects	Description	Effective Response
ra-A ontrolled Trials	81	Previously untreated	70 subjects **(86%)**
ra-A ncontrolled Trials	142	75% had been resistant or hypersensitive to IDU	101 subjects **(71%)**

TE: In the controlled and uncontrolled trials, 70 and 101 subjects, spectively, re-epithelialized at the end of three weeks on Vira-A.

Vira-A is preservative-free.

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ta on file, Medical Department, Parke-Davis

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TIMOPTIC (Timolol Maleate, MSD) alone controlled 76% of patients (485 of 638)

Based on clinical studies of 638 patients maintained on therapy from 14 to 477 days:

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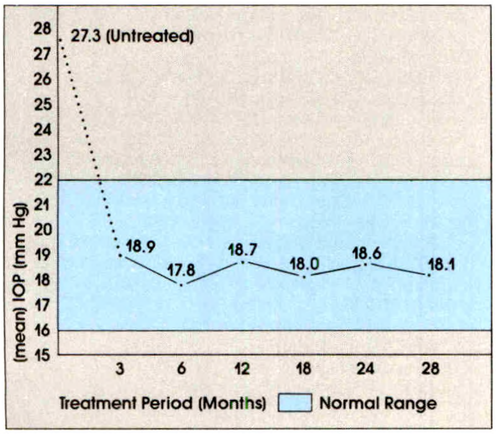
TIMOPTIC alone: highly effective long-term therapy

TIMOPTIC alone maintained control of IOP with no evidence of tolerance observed in 102 patients for over 2 years



In a long-term maintenance study, the mean IOP was reduced from an untreated level of 27.3 mm Hg to 18.9 mm Hg after three months of treatment. The mean IOP remained below this level at all subsequent intervals up to 28 months.

TIMOPTIC alone: greater patient comfort and convenience

TIMOPTIC alone offers:

- little or no change in pupil size
- little or no blurring of vision
- little or no effect on tear production
- little or no hyperemia
- no dimming of vision
- no night blindness
- simple "one-drop" b.i.d. dosage may encourage long-term compliance

There are significantly fewer and less severe side effects than with pilocarpine or epinephrine.*

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Helpful in the management of a broader range of patients.

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- In some patients who respond inadequately to maximum available drug therapy, the addition of TIMOPTIC may result in satisfactory management of intraocular pressure, thus possibly postponing the need for surgery.

TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product. Occasionally, mild ocular irritation has been reported.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma. TIMOPTIC is not recommended for use in children at this time.

*Based on controlled multiclinic studies, evaluating over 1000 patients.

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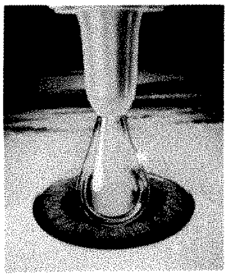

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a significant advance in the treatment of:

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- ocular hypertension
- aphakic glaucoma
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- some patients who respond inadequately to multiple antiglaucoma drug therapy

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How to transfer from current therapy to TIMOPTIC:
1. On the first day: continue with the agent(s) already being used and add one drop 0.25% TIMOPTIC in each eye b.i.d.

2. On the second day: discontinue the previously used agent(s) and continue with TIMOPTIC in each eye b.i.d.

If clinical response is not adequate:
Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC twice a day in each eye. If the intraocular pressure is maintained at satisfactory levels, the dosage schedules may be changed to one drop once a day in each eye. Dosages above one drop of 0.5% TIMOPTIC twice a day generally have not been shown to produce further reduction of IOP.

Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.

℞
Timoptic
(Timolol
Maleate | MSD)
0.25%
one drop
in each eye
b.i.d.

CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.
PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchial asthma; sinus bradycardia and greater than first-degree block; cardiogenic shock; right ventricular failure secondary to pulmonary hypertension; congestive heart failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.
Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.
Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.
ADVERSE REACTIONS: Occasionally, signs and symptoms of mild ocular irritation have been reported. Slight reduction of the resting heart rate (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed in some patients. Local hypersensitivity reactions have occurred rarely.
Note: TIMOPTIC has been used in patients with glaucoma wearing conventional (PMMA) hard contact lenses, and has generally been well tolerated. TIMOPTIC has not been studied in patients wearing lenses made with materials other than PMMA.
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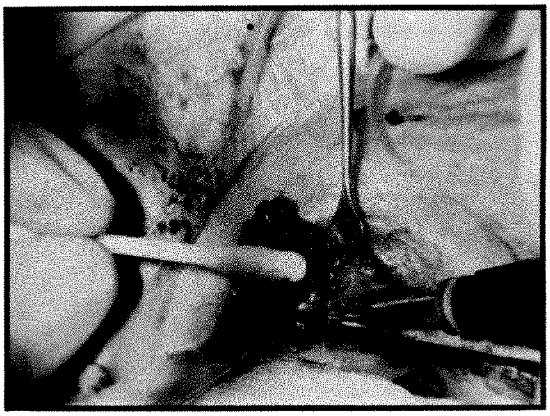

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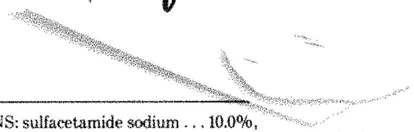

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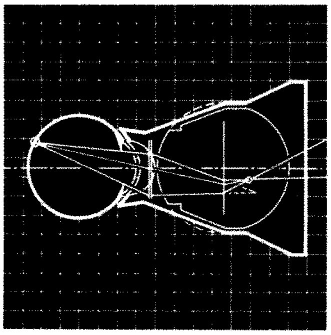

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- A written outline of the presentation
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ENROLLMENT AND CREDIT

The first program in this educational couse will be presented in July 1979. Enrollment is on a twelve-month basis with late enrollments considered retroactive to July. A written examination will be sent to enrollees in December and in June to cover the material presented during the preceding six months.

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TOPICS AND COURSE FACULTY
July 1979 — June 1980

PIGMENTED LESIONS OF THE OUTER EYE, CLINICAL ASPECTS
PAUL HENKIND, M.D., Ph.D.
Albert Einstein School of Medicine

DIAGNOSIS AND MANAGEMENT OF CONGENITAL ESOTROPIA
MARSHALL M. PARKS, M.D.
George Washington University

INTRODUCTION TO PARS PLANA VITRECTOMY
KEITH M. ZINN, M.D.
Mount Sinai School of Medicine

THE "NO TOUCH" TECHNIQUE IN OCULAR MALIGNANT MELANOMAS
F. T. FRAUNFELDER, M.D.
University of Oregon

SYNDROMES OF CHILDHOOD UVEITIS
ROBERT S. COLES, M.D.
Mount Sinai School of Medicine

MEDICAL TREATMENT OF OPEN ANGLE GLAUCOMA
THOM J. ZIMMERMAN, M.D., Ph.D.
Louisiana State University

PERIPHERAL CORNEAL ULCERS
BARTLY J. MONDINO, M.D.
University of Pittsburg

THE DIAGNOSIS AND MANAGEMENT OF ORBITAL FRACTURES
BYRON SMITH, M.D.
New York Medical College

MACULAR DISEASE
J. DONALD M. GASS, M.D.
University of Miami

DIABETIC RETINOPATHY
GEORGE H. BRESNICK, M.D.
University of Wisconsin

OCULAR HERPES SIMPLEX
DEBORAH P. LANGSTON, M.D.
Harvard Medical School

REFRACTION
BENJAMIN MILDER, M.D.
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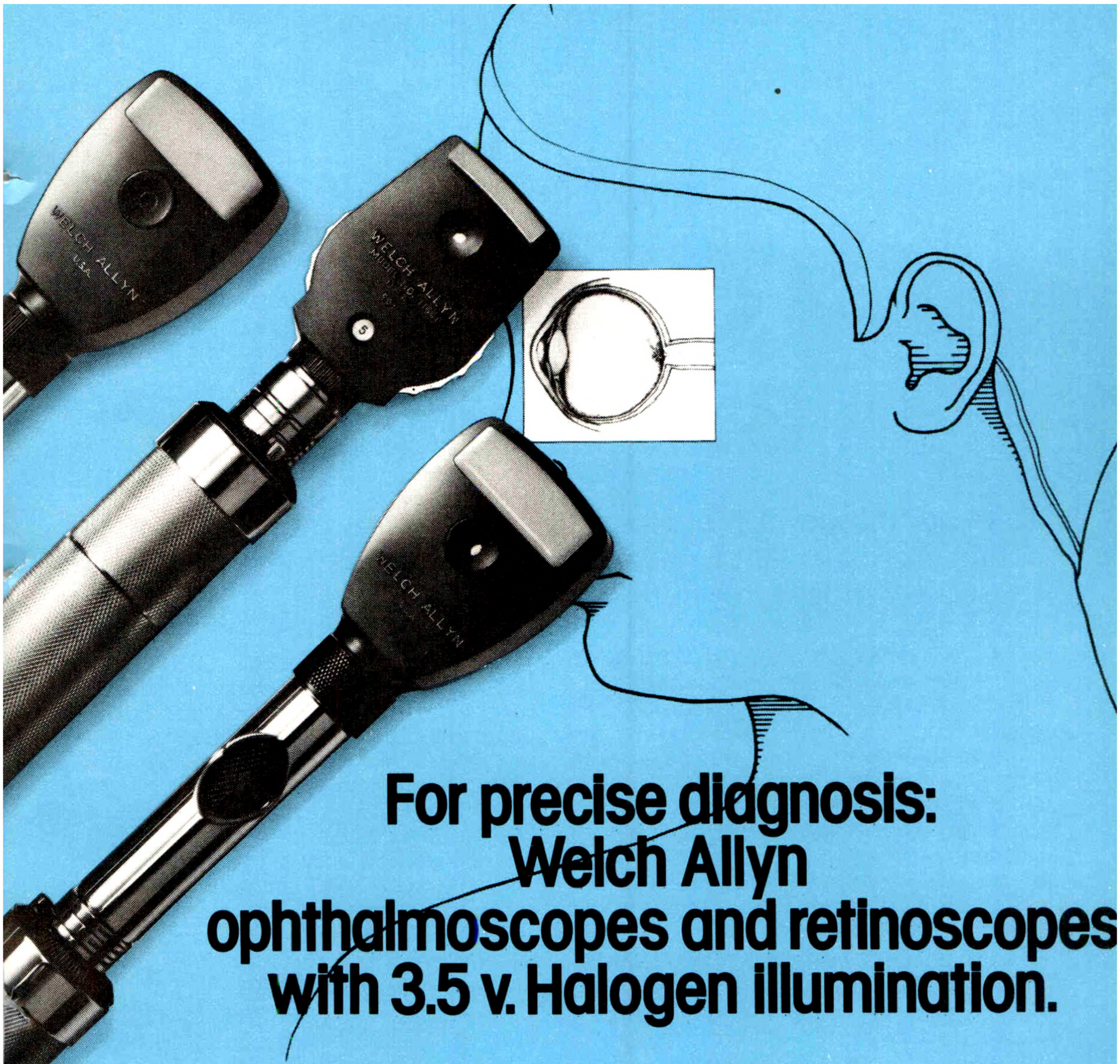

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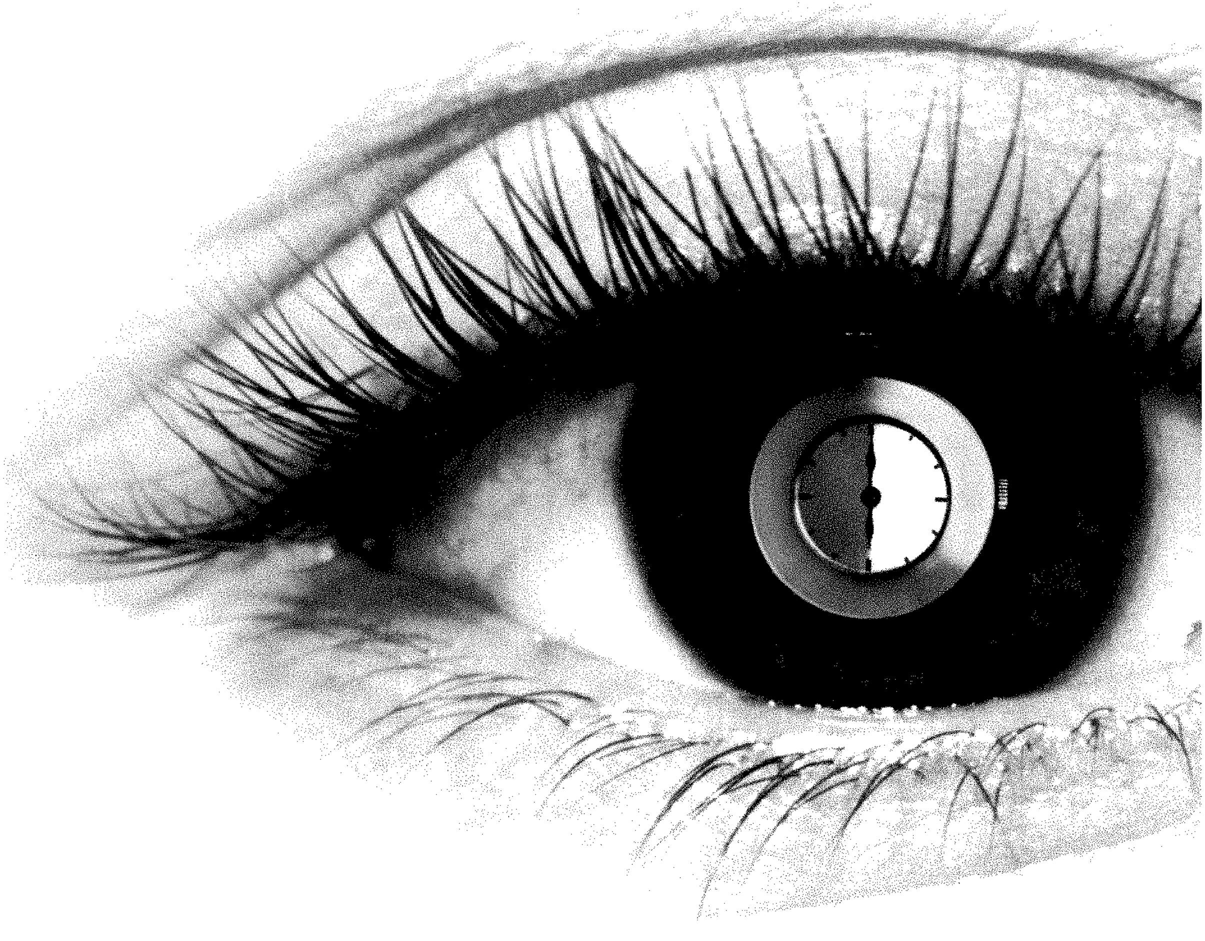

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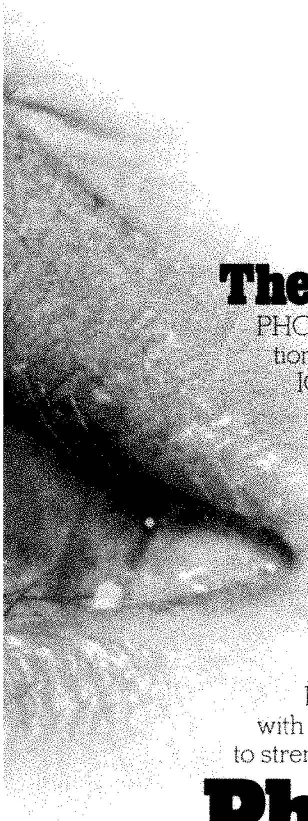

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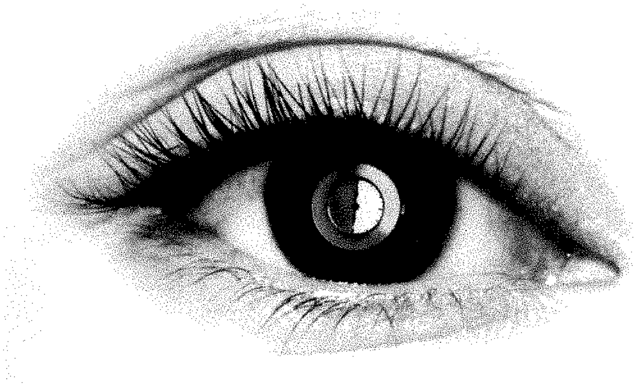

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Precautions: 1. Gonioscopy is recommended prior to initiation of therapy.

2. Where there is a quiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and ciliary muscle contraction that may occur.

3. While systemic effects are infrequent, proper use of the drug requires digital compression of the nasolacrimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

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6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer, pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.

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2. Stinging, burning, lacrimation, lid muscle twitching, conjunctival and ciliary redness, browache, induced myopia with visual blurring may occur.

3. Activation of latent iritis or uveitis may occur.

4. Iris cysts may form, and if treatment is continued, may enlarge and obscure vision. This occurrence is more frequent in children. The cysts usually shrink upon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Rarely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative esotropia.

5. Prolonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.

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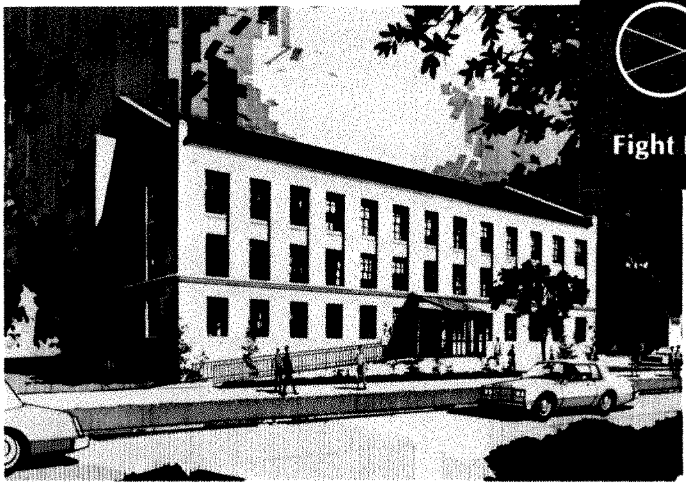

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- Frank W. Newell, M.D.
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Friday, September 14

Program — Drake Hotel, 100 E. Lake Shore Drive	8:30-5:00
Reception — Eye Research Laboratories, 939 E. 57th Street	5:00-7:00
Banquet — Quadrangle Club, 1155 E. 57th Street	7:00

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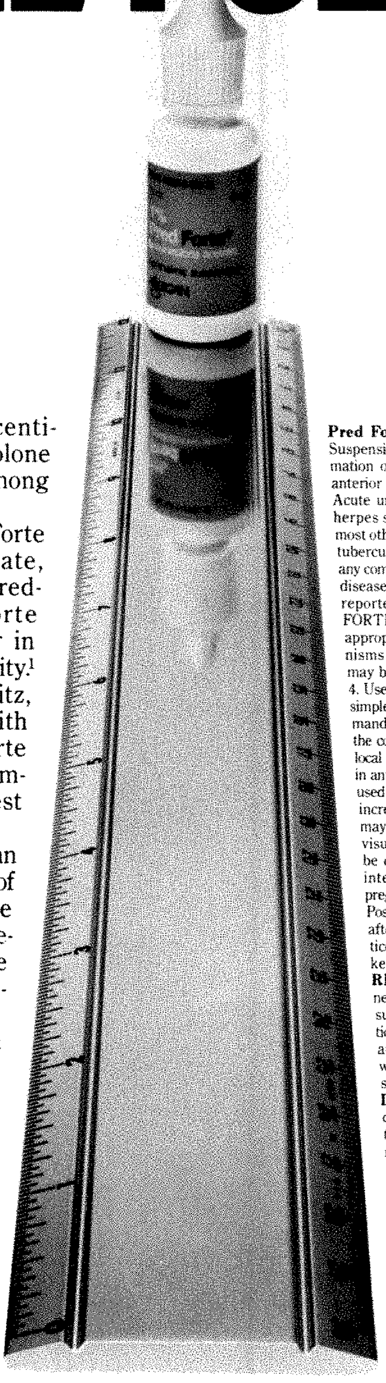

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REFERENCE NOTES: ***1.*** *Leibowitz, H. M. and Kupferman, A. Bioavailability and therapeutic effectiveness of topically administered corticosteroids. Trans Am Acad Ophthalmol Otolaryngol 79 (1): op 78-op 88, 1975.* ***2.*** *Ibid.* ***3.*** *Ibid.*

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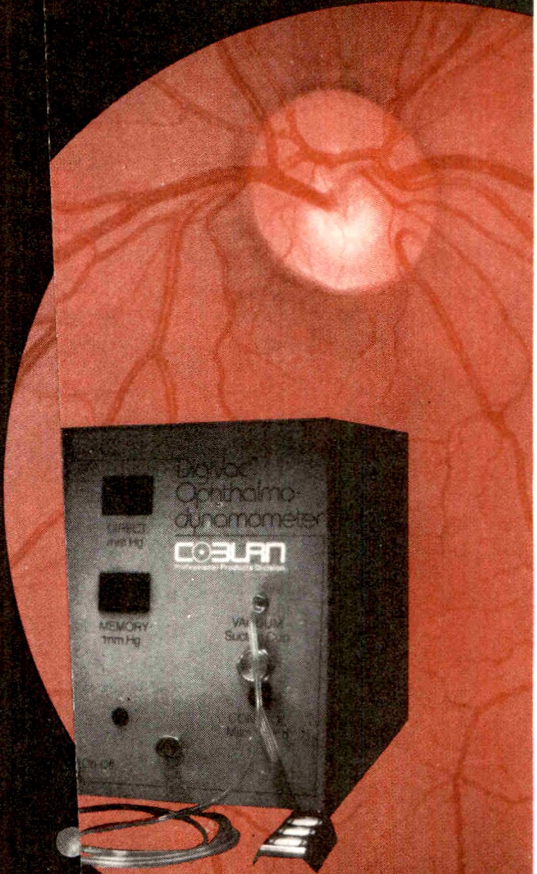

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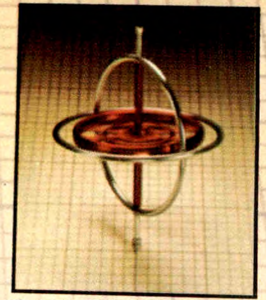

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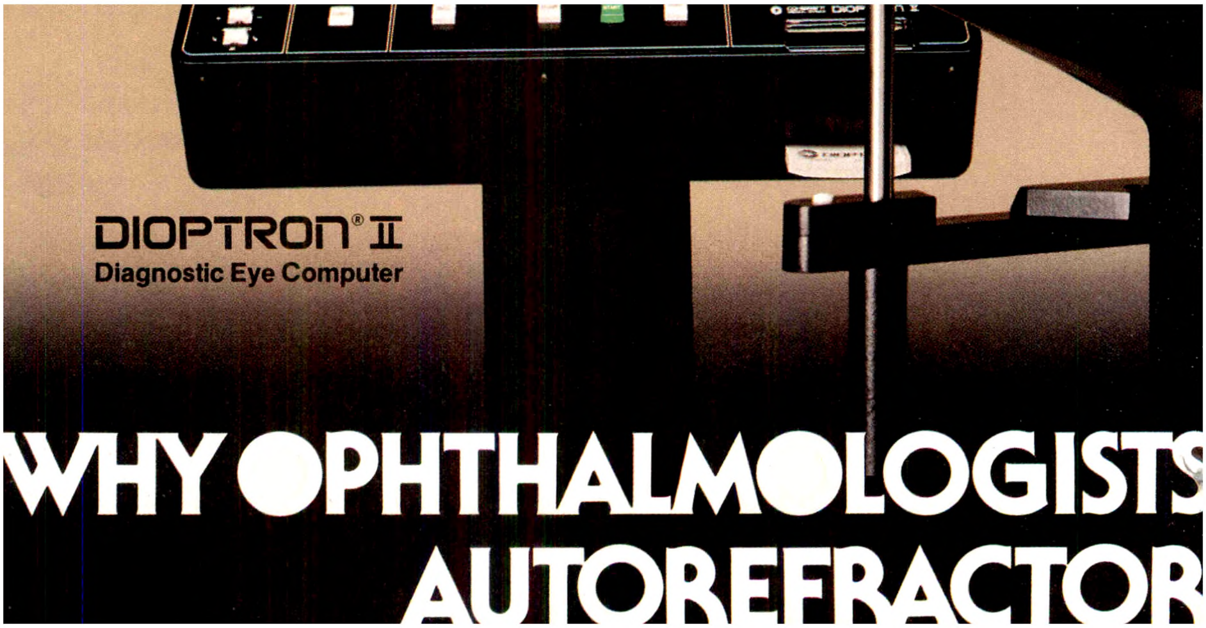
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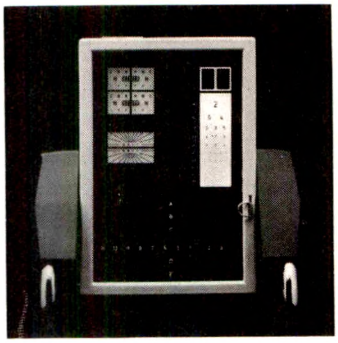

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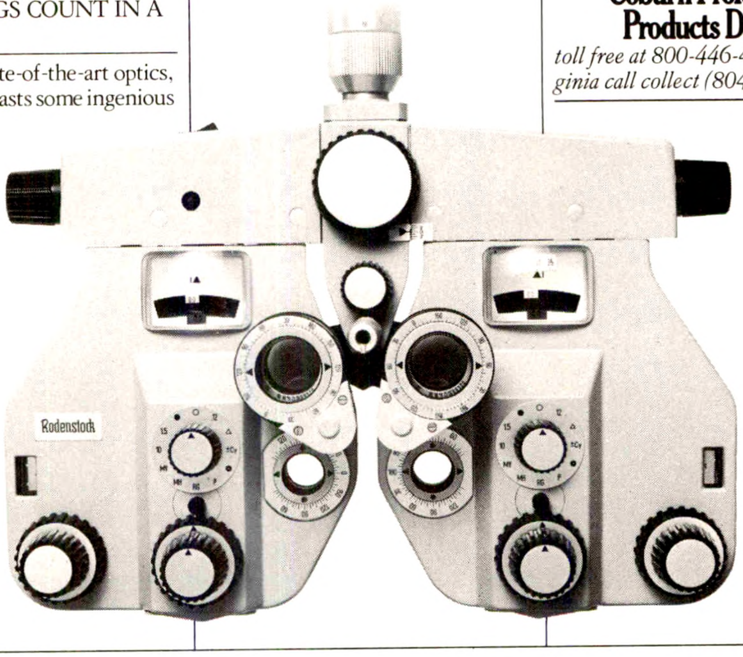

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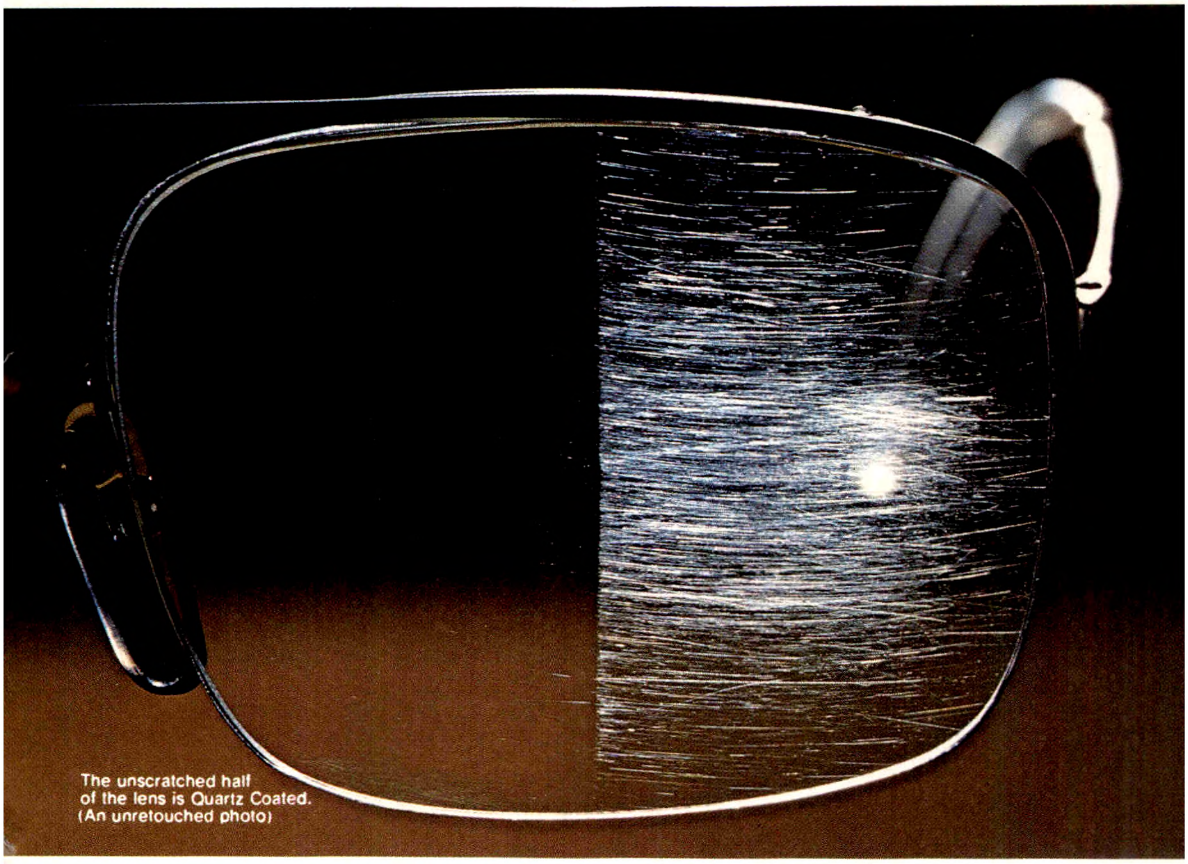

The unscratched half of the lens is Quartz Coated. (An unretouched photo)

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and others.

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J. Hagedoorn, Ph.D.
P. Janetta, M. D.
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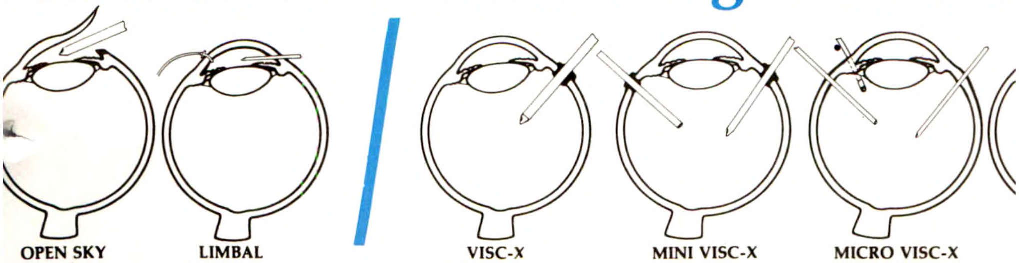

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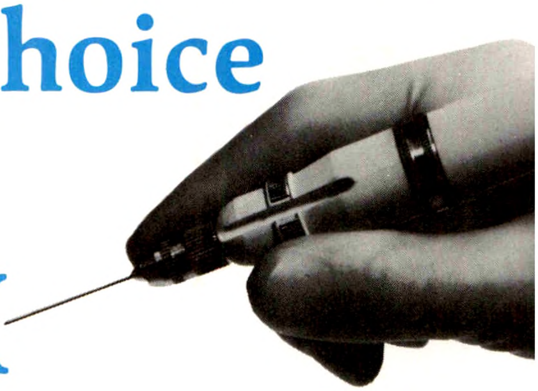

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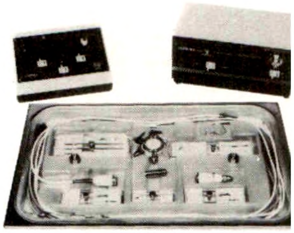

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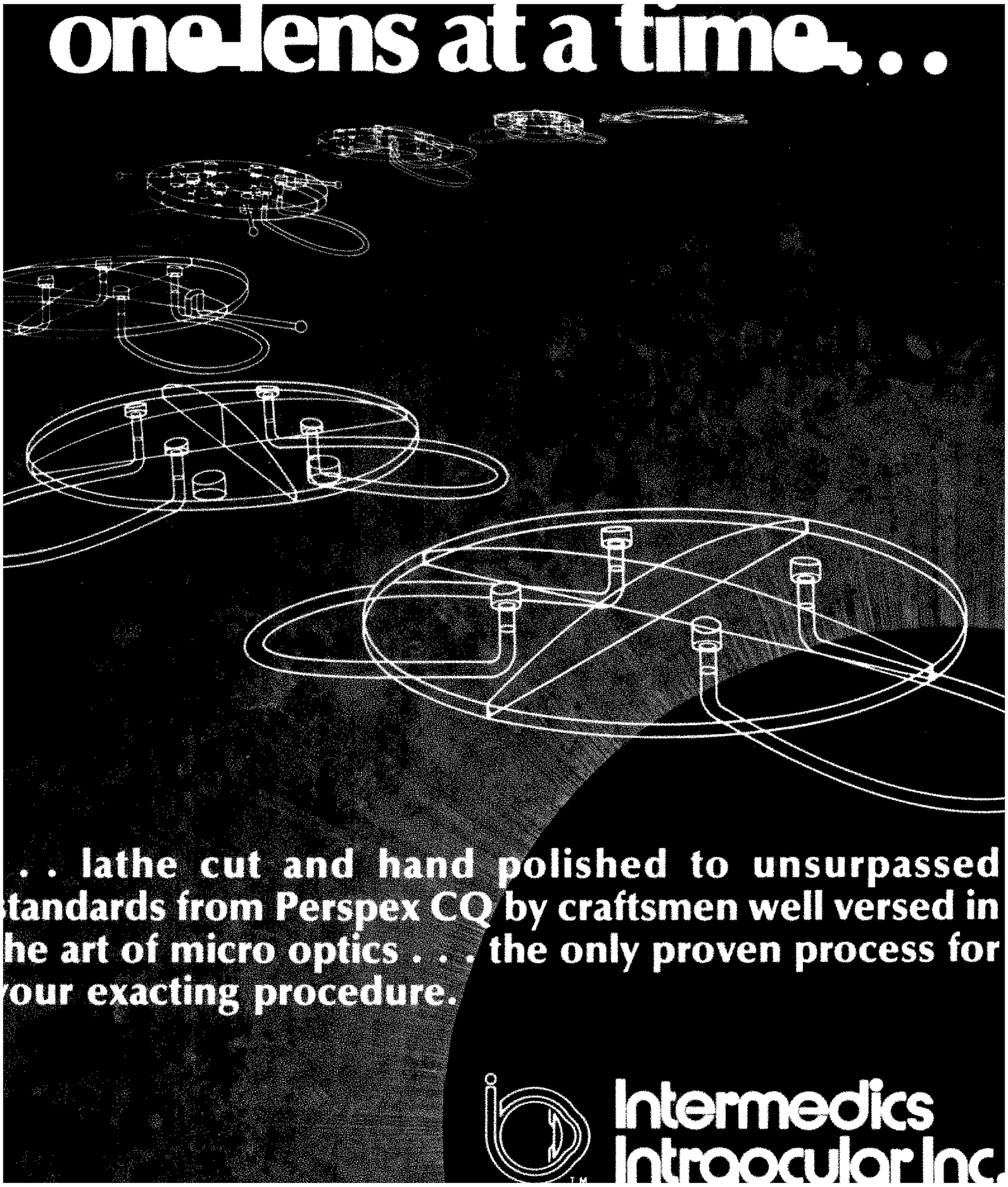

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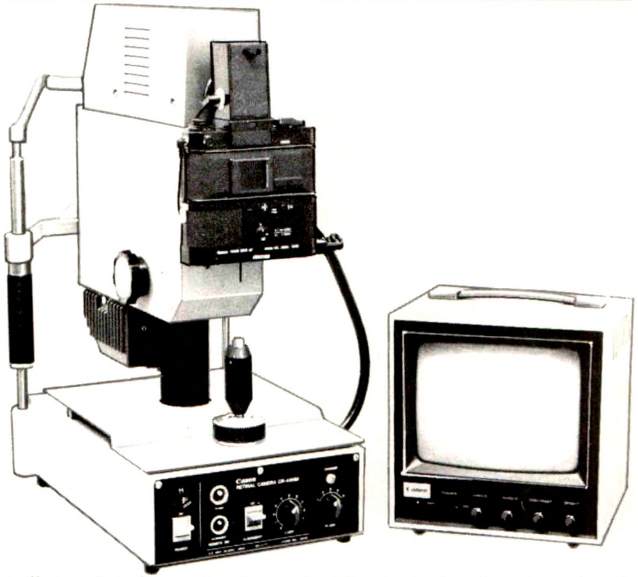

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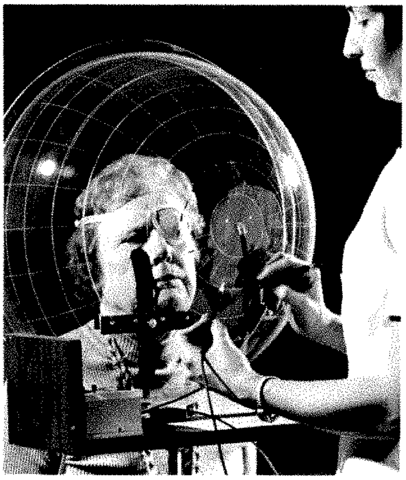

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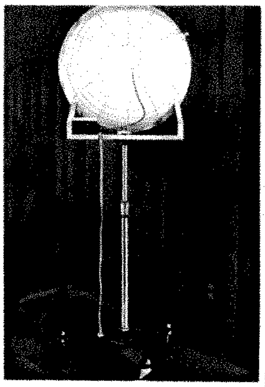

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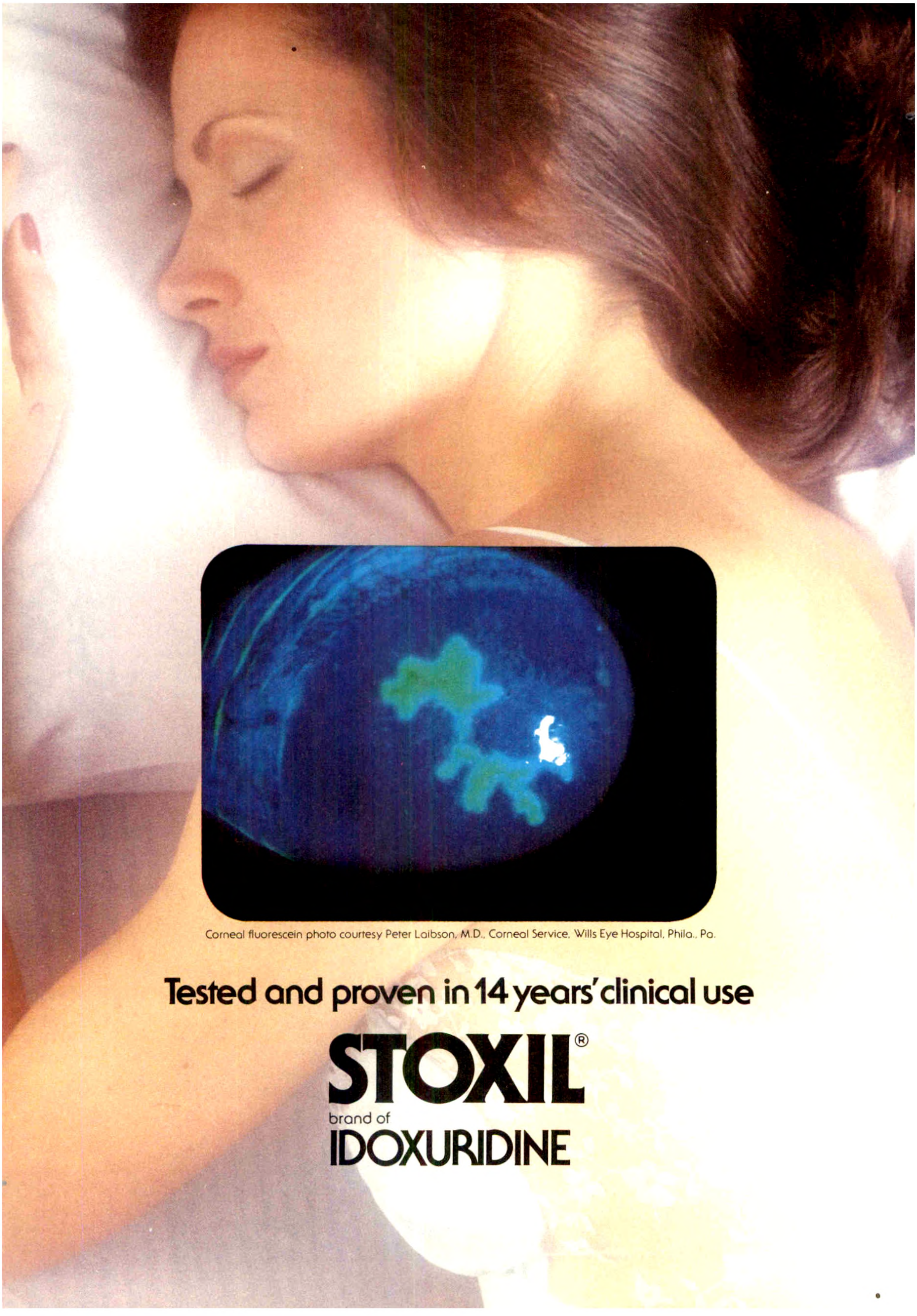

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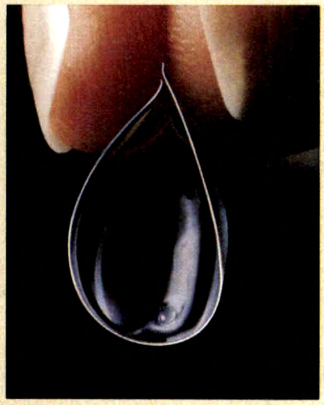
t so soft. **Now soft lenses can be worn by patients with small amounts of astigmatism.** Astigmatic patients deserve the same benefits spherical myopes enjoy with HYDROCURVE II® (bufilcon A) Contact Lenses. For patients with small amounts of astigmatism, you can now deliver results that once were attainable only with spectacles. The new specially-designed lens from HYDROCURVE Soft Lenses, Inc. enables you to easily and successfully expand your soft lens practice.



Maximum Fitting Capability by design. Maximum Fitting Capability enables you to fit more patients, more successfully, in less time. As proven in a recent study, the 13.5mm diameter 8.6mm base curve successfully fits 81% of all HYDROCURVE spherical soft lens candidates[1]; this same lens concept gives you greater fitting efficiency and excellent corneal response with HYDROCURVE II Lenses for the Correction of Small Amounts of Astigmatism. With cylinder axes ± 20° from both the horizontal and vertical meridians,[2] and a constant cylinder power of -1.25 D, the HYDROCURVE II (bufilcon A) Contact Lens can be used effectively on patients with 1.00 D to 2.00 D of with-the-rule, against-the-rule, and many oblique axes of residual astigmatism. One prism diopter provides the lens with sufficient weight differential to stabilize it on the cornea, while still maintaining a thin lens profile which optimizes corneal physiology and minimizes lid interference.

Diagnostic efficiency with a small trial set. You can determine the best lens to meet all fitting requirements without maintaining a large inventory. A four or eight lens fitting set enables you to select and evaluate the stabilization, axis and spherical over-refraction.

HYDROCURVE Soft Lenses, Inc. maintains a complete inventory of astigmatic lenses, enabling us to expedite your order and increase your practice efficiency.

HYDROCURVE Soft Lenses, Inc.—America's Contact Lens Company. HYDROCURVE Soft Lenses, Inc. is proud to introduce this new lens. Bringing you the latest innovations in contact lens technology has always been our achievement.

The thin lens profile, large optical zone, oxygen permeability and high tensile strength of HYDROCURVE Lenses for the Correction of Small Amounts of Astigmatism extend throughout the complete HYDROCURVE lens program. All to make the going soft for you.

[1] Malin, H. and Rorabaugh, D. "Fitting Small, Thin Hydrocurve Lenses." Contact Lens Forum, April, 1979.

[2] Available in 5° increments.

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8006 ENGINEER ROAD • SAN DIEGO, CALIFORNIA 92111 • (714) 277-9873

HYDROCURVE II® (bufilcon A) Contact Lenses

(bufilcon A 55% water 45%)

DESCRIPTION

The HYDROCURVE II (bufilcon A) Contact Lens is a hemispherical shell of approximately 14 mm chord diameter and 0.1 to 1.0 mm thickness. The lens material, bufilcon A, is a hydrophilic random copolymer of 2-hydroxyethyl methacrylate, N-(1,1-Dimethyl-3-oxobutyl)-acrylamide, and methacrylic acid. The polymer is a 3-dimensional network of copolymer chains joined by trimethylolpropane trimethacrylate crosslinks at a density of about one crosslink for every 1400 monomer units. It consists of 55% bufilcon A and 45% water when immersed in normal saline.

ACTIONS

In its hydrated state a HYDROCURVE II Contact Lens becomes soft and pliable. When placed on the human cornea the hydrated lens acts as a refracting medium to compensate spherical ametropias. The material has a refractive index of 1.43.

INDICATIONS

HYDROCURVE II Contact Lenses are indicated for the correction of visual acuity in persons with non-diseased eyes who have spherical ametropias, refractive astigmatism of 1.50 diopters or less and/or corneal astigmatism of 2.00 diopters or less and aphakia.

CONTRAINDICATIONS

HYDROCURVE II Contact Lenses are contraindicated by the presence of any of the following conditions:

1. Acute and subacute inflammations of the anterior segment of the eye.
2. Any eye disease which affects the cornea or conjunctiva.
3. Insufficiency of lacrimal secretion.
4. Corneal hypoesthesia.
5. Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Medicaments and Eye Drops
The HYDROCURVE II Contact Lens must be stored ONLY in BOILnSOAK® Sterile Preserved Saline Storage Solution or FLEXSOL® Sterile Solution for Disinfection and Storage. No ophthalmic solutions or medicaments, including conventional hard contact lens solutions, can be used by HYDROCURVE II Contact Lens wearers prior to or while the lens is in place on the eye. Only the recommended disinfectant soaking solutions BOILnSOAK or FLEXSOL, the recommended cleaners SOFT MATE® or PREFLEX®, NORMOL® Rinsing Solution or the recommended lubricating solution ADAPETTES® may be used on the HYDROCURVE II Contact Lens.

Abrasions and Infections
If the lens becomes less comfortable to the wearer than when it was first placed on the wearer's cornea, this may indicate the presence of a foreign body. The lens should be removed immediately and the patient examined. If any corneal abrasion, ulceration, irritation or infection is present, a physician should be consulted immediately.

Aphakic Patients
Aphakic patients should not be fitted with HYDROCURVE II Contact Lenses during the postoperative period until, in the opinion of the surgeon, the eye has healed completely.

Lens Care Regimen
Patients must adhere to the recommended daily lens care regimen of HYDROCURVE II Contact Lenses. Failure to follow this procedure may result in development of serious ocular infections.

Wearing Restrictions
The HYDROCURVE II Contact Lens should be removed before sleeping or swimming or in the presence of noxious and irritating vapors.

Visual Blurring
When visual blurring occurs the lens must be removed until the condition subsides.

PRECAUTIONS

Storage
HYDROCURVE II Contact Lenses must be stored ONLY in BOILnSOAK or FLEXSOL Solution. If left exposed to air, the lenses will dehydrate. If a lens dehydrates, it should be soaked ONLY in BOILnSOAK or FLEXSOL Solution until it returns to a soft, supple state.

Cleaning and Disinfection
HYDROCURVE II Contact Lenses must be BOTH cleaned and disinfected daily. One procedure does not replace the other. CLEANING is necessary to remove mucus and film from the lens surface. This can be accomplished by using either SOFT MATE or PREFLEX on a daily basis. Excessive deposits may damage the lens; therefore, if this occurs, evaluation of the lens care procedures should be made.

DISINFECTION with a HYDROCURVE Patient Disinfection Unit and BOILnSOAK Solution has been tested microbiologically and shown to be an effective disinfection procedure for HYDROCURVE II Contact Lenses.

HYDROCURVE II Contact Lenses must be cleaned daily with SOFT MATE or PREFLEX. Fresh BOILnSOAK Solution must be used daily for storing the lenses. The HYDROCURVE Carrying Case must be emptied and refilled with fresh BOILnSOAK Solution each time the lens is stored.

If a HYDROCURVE Patient Disinfection Unit is not available for disinfection of the lenses, the lenses must be boiled in their Carrying Case in a pan of water for 10 minutes.

CHEMICAL DISINFECTION with FLEXSOL has been tested microbiologically and shown to be an effective disinfection procedure for HYDROCURVE II Contact Lenses. HYDROCURVE II Contact Lenses must be cleaned and rinsed daily, or after wearing, with PREFLEX and NORMOL. The HYDROCURVE Twin Case must be emptied and refilled with fresh FLEXSOL prior to disinfecting the lenses. Fresh FLEXSOL must be used daily for storing and disinfecting the lenses.

WARNING
DO NOT MIX OR ALTERNATE THE DISINFECTION AND STORAGE SYSTEMS. FLEXSOL SHOULD NOT BE USED WITH HEAT.

Hygiene
Patients must wash and rinse hands thoroughly, and dry with a lint-free towel before handling the lenses.

Cosmetics, lotions, soaps and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Fluorescein
Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with normal saline solution and wait at least one hour before replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescein irreversibly.

ADVERSE REACTIONS

Serious corneal damage may result from wearing a lens which has been soaked in a conventional hard contact lens solution containing preservatives which should not be used with soft hydrophilic contact lenses.

Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritation.

Very rarely a lens may adhere to an eye as a result of the patient sleeping while wearing the lens, or wearing a hypotonic lens. If a lens adheres for any reason, patients may be instructed to apply a few drops of BOILnSOAK Solution or ADAPETTES and wait until the lens moves freely before removing the lens.

Rainbows or halos around objects or blurring of the vision may occur if the lenses are worn continuously for too long a time. Removal of the lenses and a rest period of at least one hour generally relieves these symptoms.

Excessive tearing, unusual eye secretions, and photophobia are not normal; if these symptoms occur, the patient should be examined to determine their cause.

DOSAGE and ADMINISTRATION

Conventional methods of fitting contact lenses do not apply to HYDROCURVE II (bufilcon A) Contact Lenses. For a detailed description of the fitting technique, refer to the HYDROCURVE Fitting Guide, copies of which are available from:

Soft Lenses, Inc.
8006 Engineer Road
San Diego, California 92111

WEARING SCHEDULE SHOULD BE DETERMINED BY THE EYE CARE PRACTITIONER. STUDIES HAVE NOT BEEN PERFORMED TO SUPPORT SAFETY FOR EXTENDED WEAR OF THIS LENS.

	Maximum Wearing Time
Day	(Continuous Hours)
1	3
2	3
3	4
4	4
5	6
6	6
7	8

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Patient Instructions

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  Case ....
Patient Instructions

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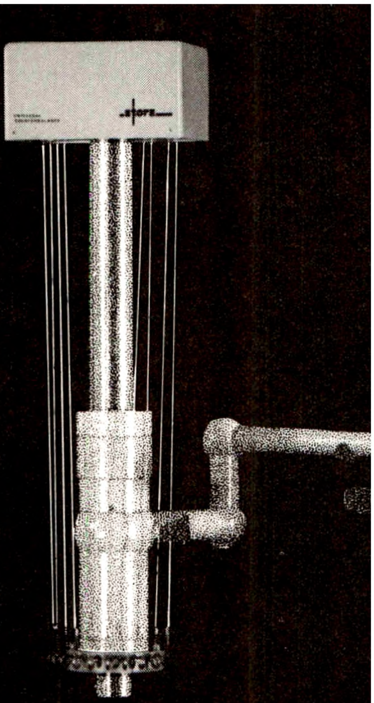

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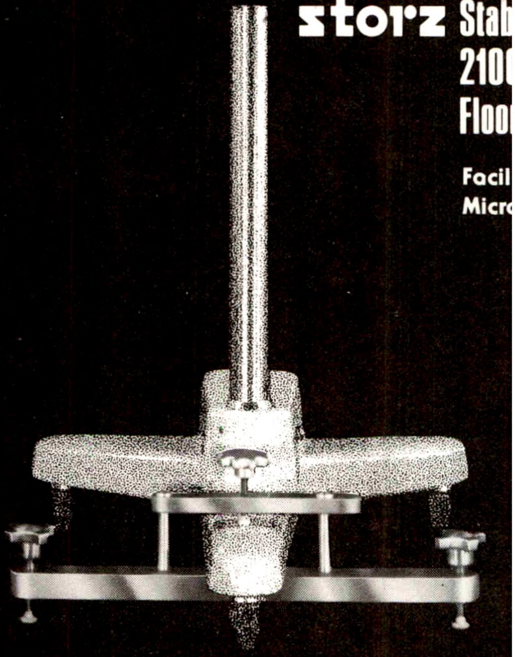

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for accommodative esotropia

The emotional impact of accommodative esotropia on a young child need not necessarily be followed by the trauma of surgery or the inconvenience and problems associated with wearing and caring for prescription lenses. The agent frequently used in the diagnosis of the condition is the same agent that can be used to correct the accommodative factor without inconvenience to the young patient.

In diagnosis... One drop of PHOSPHOLINE IODIDE 0.125% instilled daily in each eye prior to retiring, for two or three weeks, will help to determine if there is an accommodative basis for the esotropia.

In treatment... If there is a significant accommodative factor present, the continued use of PHOSPHOLINE IODIDE alone is often sufficient to correct the problem, as long as the drug is well tolerated. PHOSPHOLINE IODIDE acts by altering the accommodative convergence/accommodation relationship in a favorable way, so that near vision is obtained with less accommodative effort and fusion can frequently be reestablished.

If corrective lenses are necessary, PHOSPHOLINE IODIDE may permit the use of single vision lenses instead of bifocals.

If surgery is necessary, postoperative use of PHOSPHOLINE IODIDE may help correct a residual deviation.

BRIEF SUMMARY

(For full prescribing information, see package circular.)

PHOSPHOLINE IODIDE®

(ECHOTHIOPHATE IODIDE FOR OPHTHALMIC SOLUTION)

PHOSPHOLINE IODIDE is a long-acting cholinesterase inhibitor for topical use.

Indications: Glaucoma – Chronic open-angle glaucoma. Subacute or chronic angle-closure glaucoma after iridectomy or where surgery is refused or contraindicated. Certain non-uveitic secondary types of glaucoma, especially glaucoma following cataract surgery.

Accommodative esotropia – Concomitant esotropias with a significant accommodative component.

Contraindications: 1. Active uveal inflammation.

2. Most cases of angle-closure glaucoma, due to the possibility of increasing angle block.

3. Hypersensitivity to the active or inactive ingredients.

Warnings: 1. *Use in Pregnancy:* Safe use of anticholinesterase medications during pregnancy has not been established, nor has the absence of adverse effects on the fetus or on the respiration of the neonate.

2. Succinylcholine should be administered only with great caution, if at all, prior to or during general anesthesia to patients receiving anticholinesterase medication because of possible respiratory or cardiovascular collapse.

3. Caution should be observed in treating glaucoma with PHOSPHOLINE IODIDE in patients who are at the same time undergoing treatment with systemic anticholinesterase medications for myasthenia gravis, because of possible adverse additive effects.

Precautions: 1. Gonioscopy is recommended prior to initiation of therapy.

2. Where there is a quiescent uveitis or a history of this condition, anticholinesterase therapy should be avoided or used cautiously because of the intense and persistent miosis and ciliary muscle contraction that may occur.

3. While systemic effects are infrequent, proper use of the drug requires digital compression of the nasolacrimal ducts for a minute or two following instillation to minimize drainage into the nasal chamber with its extensive absorption area. The hands should be washed immediately following instillation.

4. Temporary discontinuance of medication is necessary if salivation, urinary incontinence, diarrhea, profuse sweating, muscle weakness, respiratory difficulties, or cardiac irregularities occur.

5. Patients receiving PHOSPHOLINE IODIDE who are exposed to carbamate or organophosphate type insecticides and pesticides (professional gardeners, farmers, workers in plants manufacturing or formulating such products, etc.) should be warned of the additive systemic effects possible from absorption of the pesticide through the respiratory tract or skin. During periods of exposure to such pesticides, the wearing of respiratory masks, and frequent washing and clothing changes may be advisable.

6. Anticholinesterase drugs should be used with extreme caution, if at all, in patients with marked vagotonia, bronchial asthma, spastic gastrointestinal disturbances, peptic ulcer, pronounced bradycardia and hypotension, recent myocardial infarction, epilepsy, parkinsonism, and other disorders that may respond adversely to vagotonic effects.

7. Anticholinesterase drugs should be employed prior to ophthalmic surgery only as a considered risk because of the possible occurrence of hyphema.

8. PHOSPHOLINE IODIDE (echothiophate iodide) should be used with great caution, if at all, where there is a prior history of retinal detachment.

Adverse Reactions: 1. Although the relationship, if any, of retinal detachment to the administration of PHOSPHOLINE IODIDE has not been established, retinal detachment has been reported in a few cases during the use of PHOSPHOLINE IODIDE in adult patients without a previous history of this disorder.

2. Stinging, burning, lacrimation, lid muscle twitching, conjunctival and ciliary redness, browache, induced myopia with visual blurring may occur.

3. Activation of latent iritis or uveitis may occur.

4. Iris cysts may form, and if treatment is continued, may enlarge and obscure vision. This occurrence is more frequent in children. The cysts usually shrink upon discontinuance of the medication, reduction in strength of the drops or frequency of instillation. Rarely, they may rupture or break free into the aqueous. Regular examinations are advisable when the drug is being prescribed for the treatment of accommodative esotropia.

5. Prolonged use may cause conjunctival thickening, obstruction of nasolacrimal canals.

6. Lens opacities occurring in patients under treatment for glaucoma with PHOSPHOLINE IODIDE have been reported and similar changes have been produced experimentally in normal monkeys. Routine examinations should accompany clinical use of the drug.

7. Paradoxical increase in intraocular pressure may follow anticholinesterase instillation. This may be alleviated by prescribing a sympathomimetic mydriatic such as phenylephrine.

Overdosage: Antidotes are atropine, 2 mg parenterally; PROTOPAM® CHLORIDE (pralidoxime chloride), 25 mg per kg intravenously; artificial respiration should be given if necessary.

How Supplied: Four potencies are available. 1.5 mg package for dispensing 0.03% solution; 3.0 mg package for 0.06% solution; 6.25 mg package for 0.125% solution; 12.5 mg package for 0.25% solution. Also contains potassium acetate (sodium hydroxide or acetic acid may have been incorporated to adjust pH during manufacturing), chorobutanol (chloral derivative), mannitol, boric acid and exsiccated sodium phosphate.

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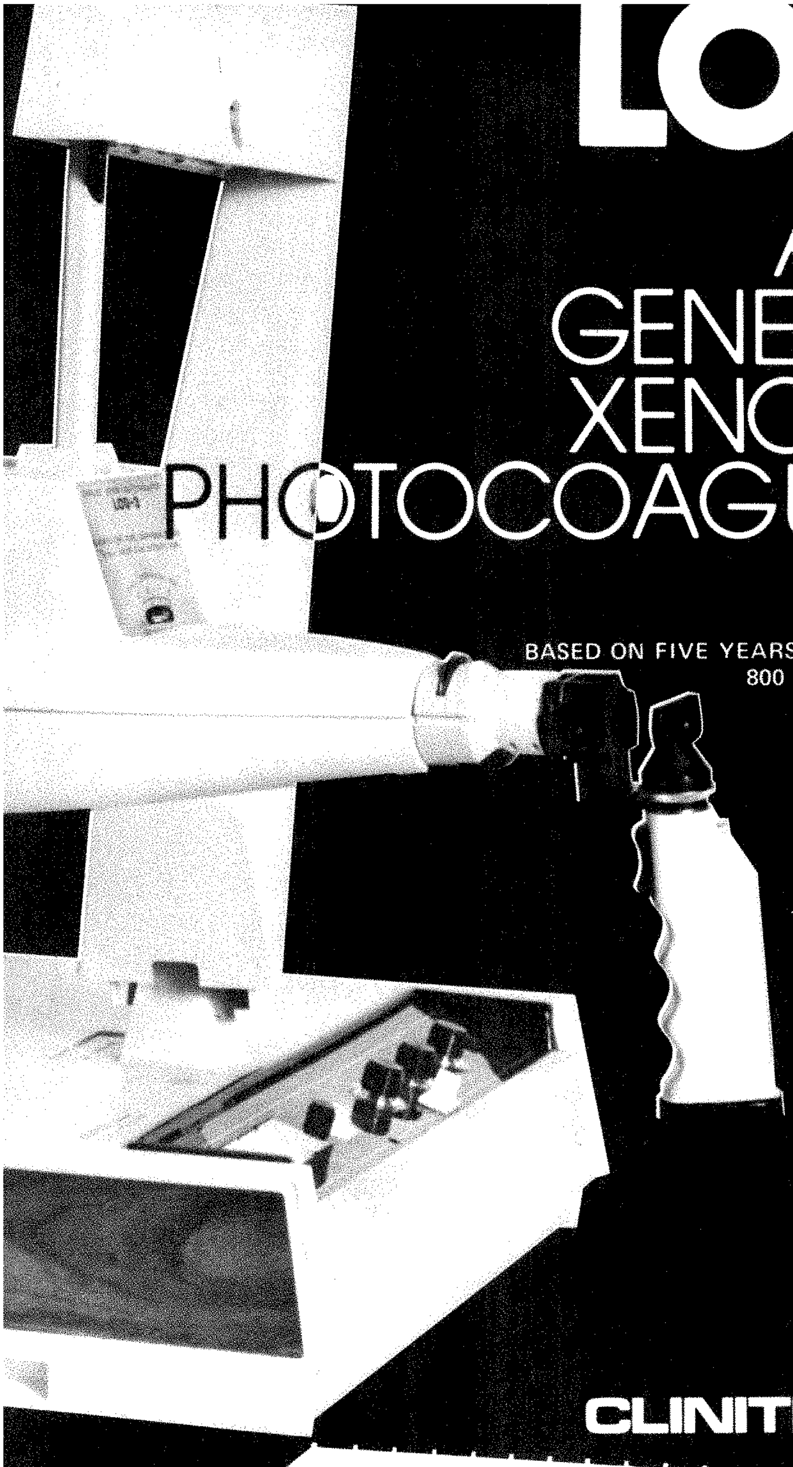

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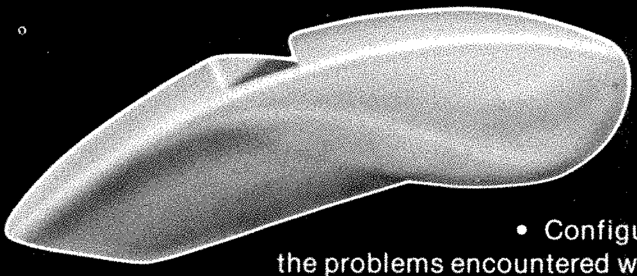

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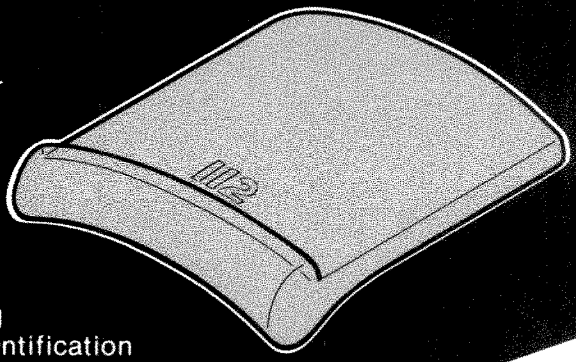

Both implants have an engraved catalog number on their surface for positive identification and proven Medical Instrument Research Associates, Inc. quality backed by nearly twenty years of clinical experience.

* The Fishmouth Phenomenon II. Wedge Scleral Buckling. Arch. Ophthalmol. Vol. 95: 1782-1787, Oct. 1977.

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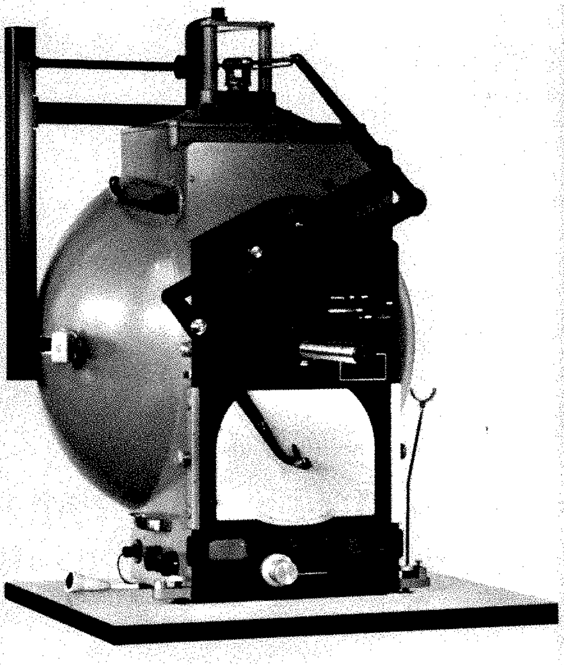

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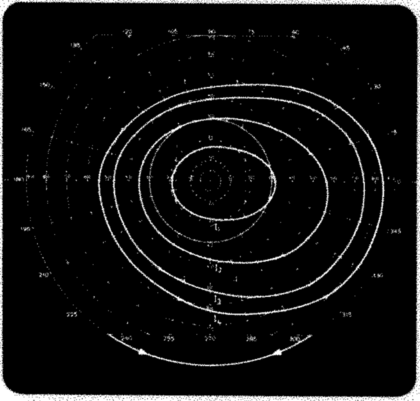

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Dates	Locations	Director	Workshop Coordinator
Jul. 26, 27	Resorts International Hotel Atlantic City, NJ	Alexander J. Brucker, M.D.	Donna Wengert (215) 662-8675
Aug. 10, 11	Abbott-Northwestern Hospital Minneapolis, Minn	Donald P. LeWin, M.D.	Nancy Peterson (612) 874-5212
Aug. 24, 25	St. Lukes Hospital Cleveland, Ohio	L. J. Singerman, M.D. Howard Kohn, M.D.	Charleen Hanford (216) 421-1213
Sep. 20, 21	Emory Univ. Hospital Atlanta, GA	Frank Bell, M.D.	Mrs. Bruni Finnerty (404) 321-0111, Ex 433
Oct. 5, 6	St. Johns Hospital Detroit, Mich	Howard Joondeph, M.D.	Mrs. Marion Lynch (313) 824-4800, Ex 208
Oct. 26, 27	Santa Ana Tustin Comm. Hosp. Santa Ana, CA (L.A. area)	John Maggiano, M.D.	Ms. Joanne Silva (714) 534-8373
Dec. 7, 8	St. Joseph's Hospital Chicago, IL	Charles Vygantis, M.D.	Becky Wilde (312) 871-8444

Information on the Workshop schedule for 1980 can be obtained from Berkeley Bio-Engineering.

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Pediatric Ophthalmology and Strabismus Update

October 4, 5 and 6, 1979

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Howard Eggers, M.D.
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Max Forbes, M.D.
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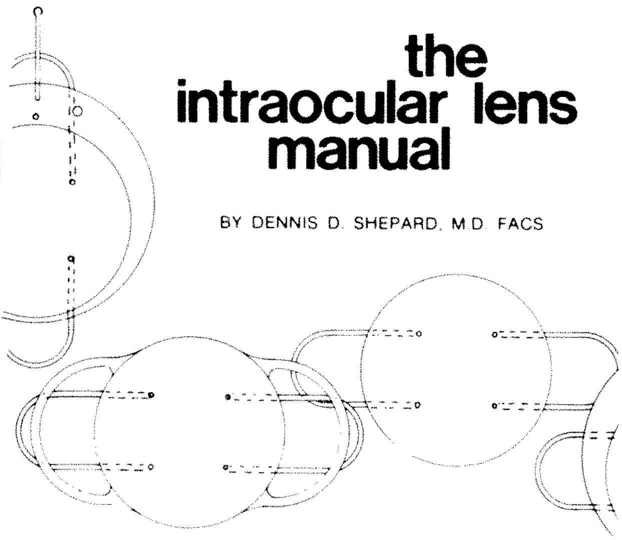

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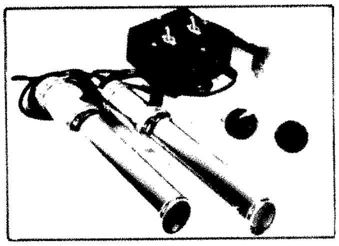

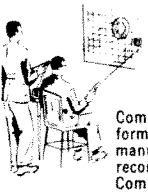

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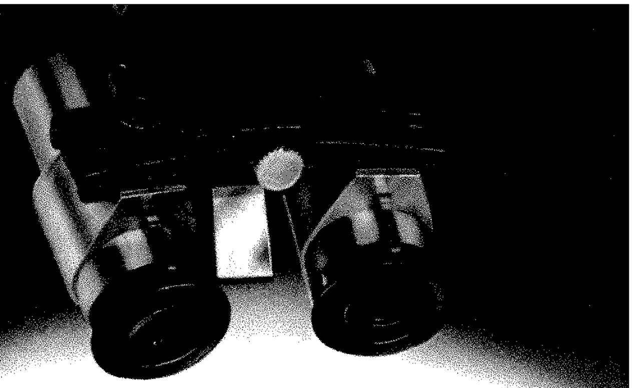

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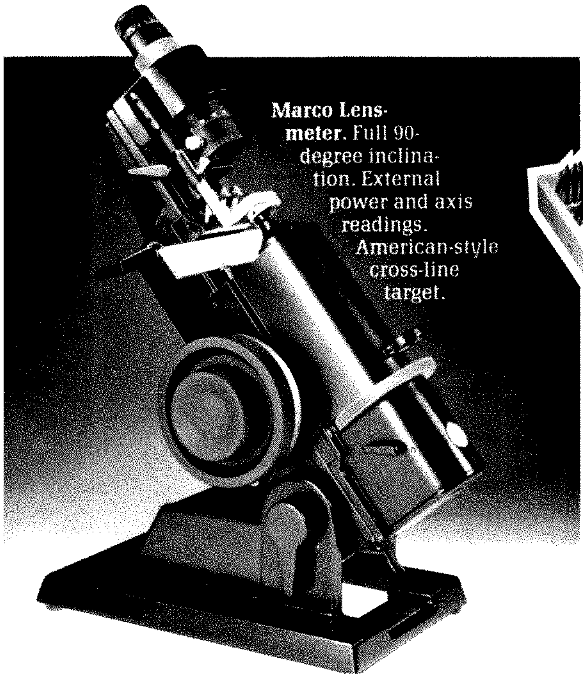

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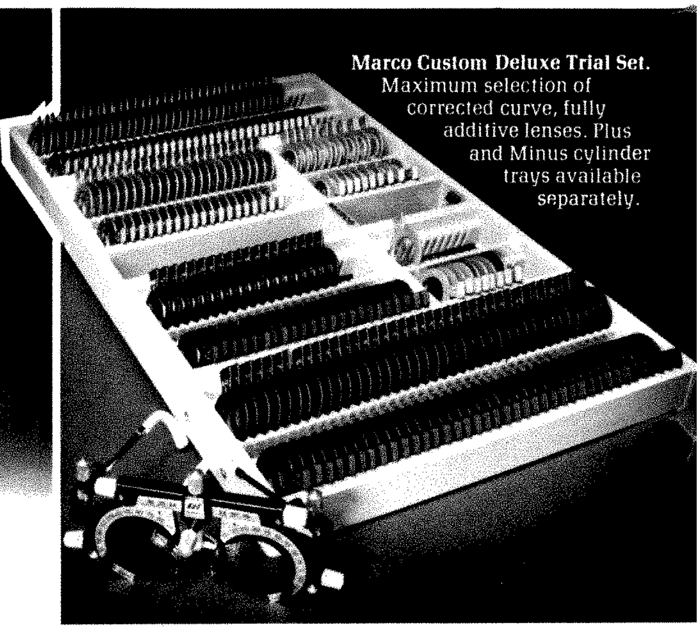

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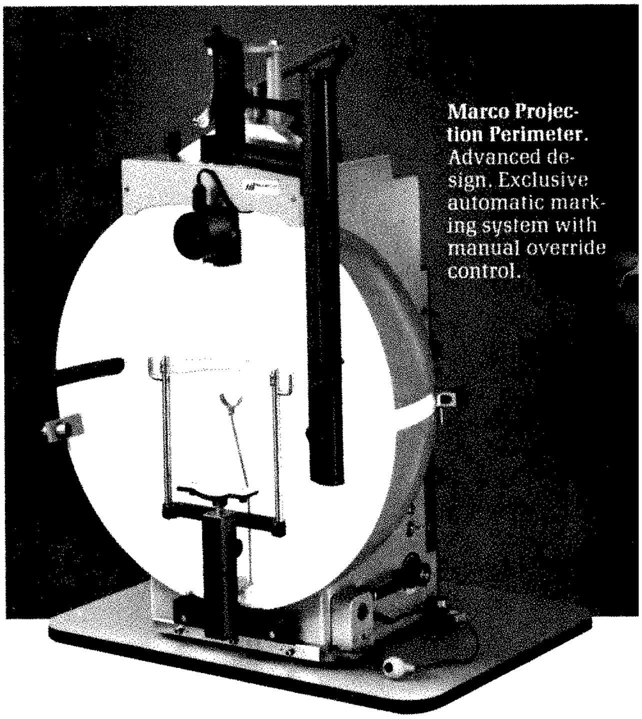

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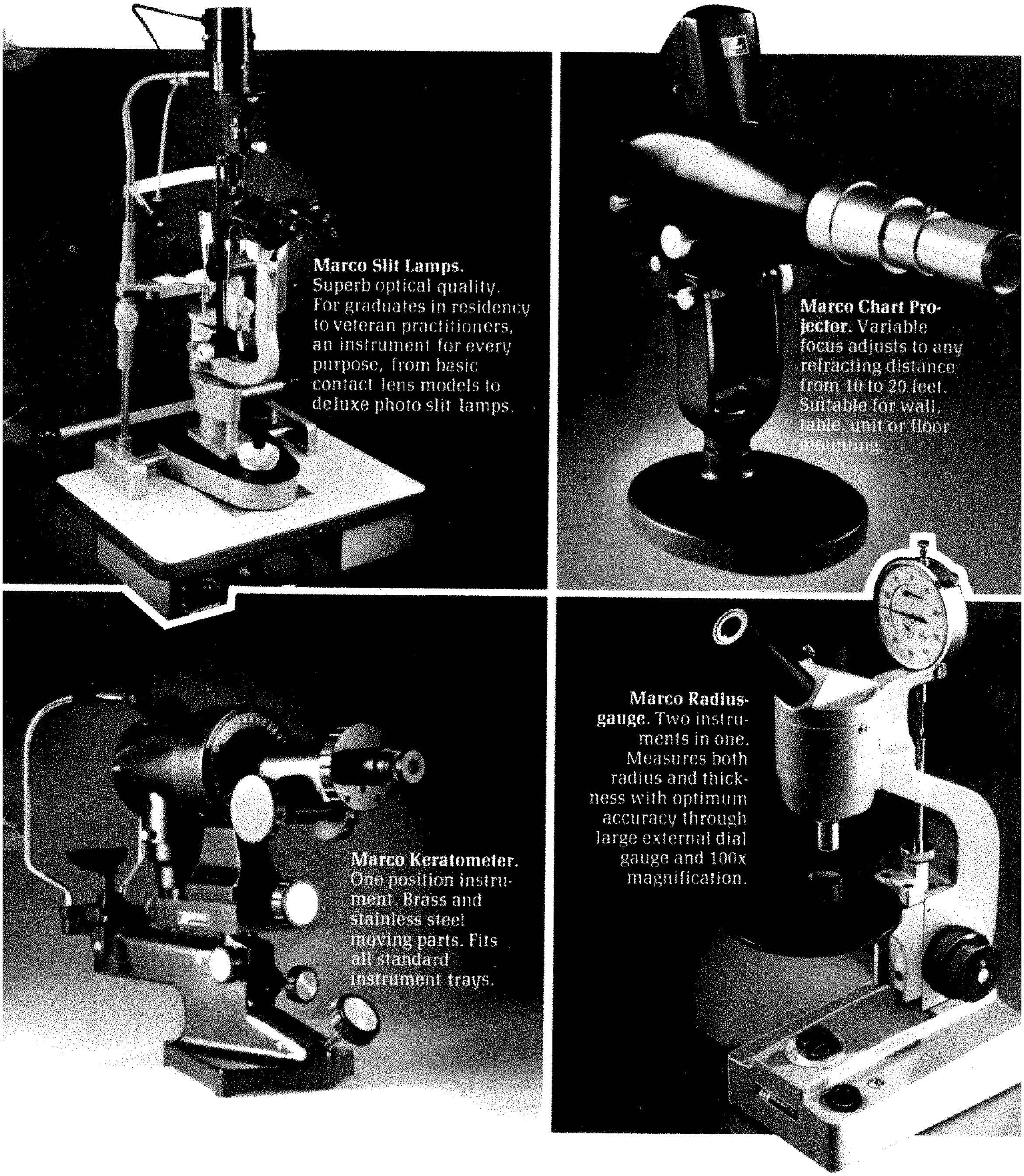

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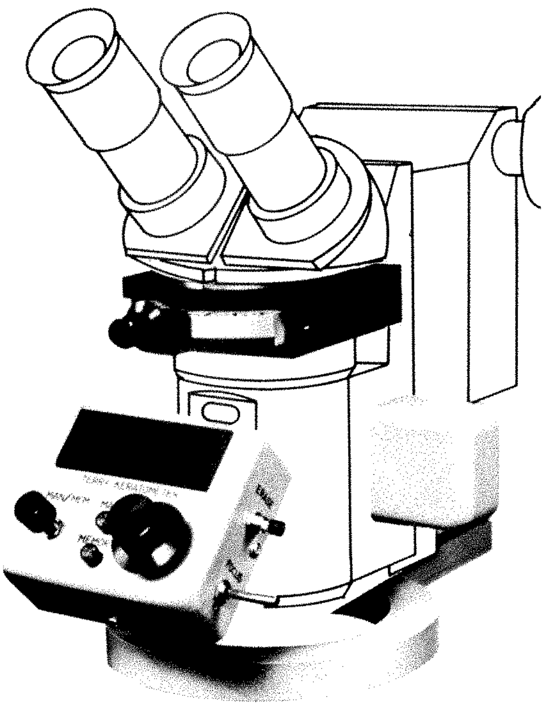

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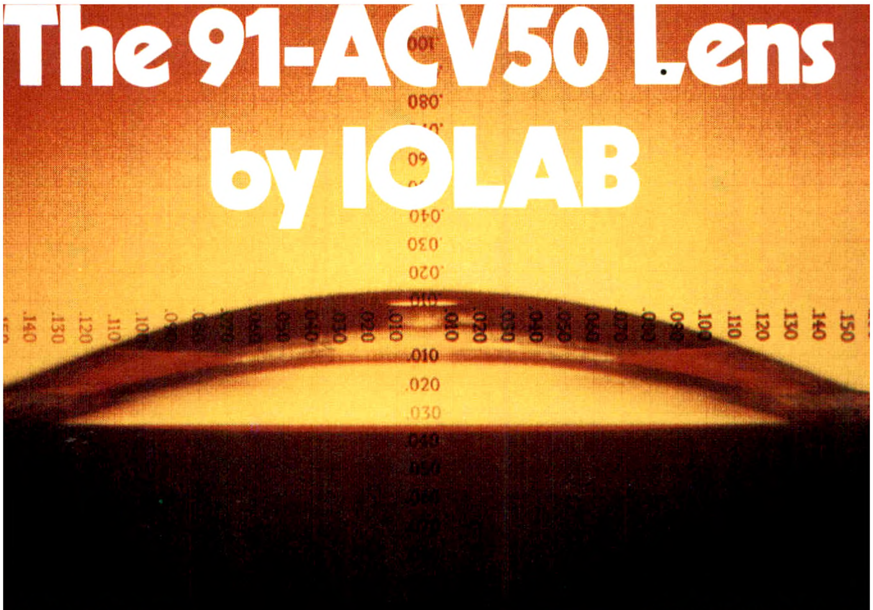

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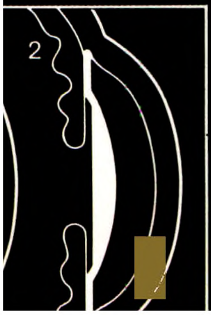

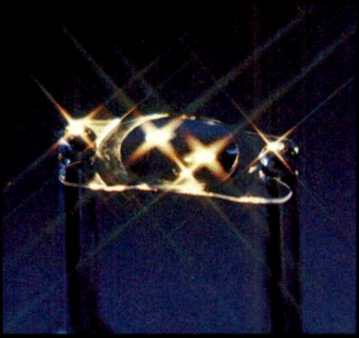

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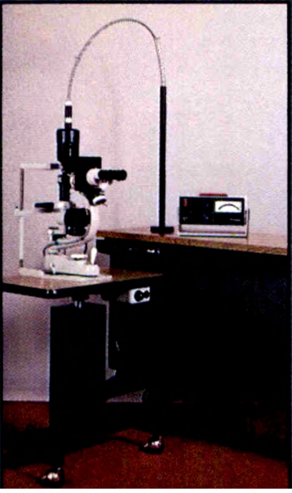

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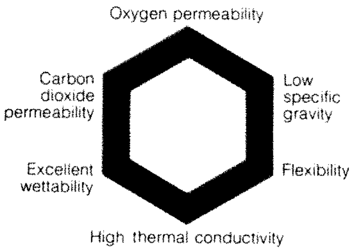

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ADVERSE REACTIONS:
As with any contact lens, corneal edema and corneal abrasions may occur if the lens is not properly fitted, or if the wearing time is excessive.

Excessive tearing, unusual eye secretions and excessive sensitivity to light are not normal; if these symptoms occur, the patient should be examined by an eye-care practitioner to determine their cause.

WARNINGS:
The RX-56® (porofocon A) contact lens must be stored only in Flexsol® solution.

No other storage solution is recommended. Patients must adhere to the recommended daily care regimen.
FAILURE TO DO SO MAY RESULT IN OCULAR INFECTION.

If the lens becomes more uncomfortable to the wearer than when first placed on the eye, the lens should be removed, checked for dirt or scratches, and cleaned. If this condition persists or if the eye becomes red or painful, the lens should be removed immediately. If symptoms persist, your eye-care practitioner should be consulted.

The RX-56® (porofocon A) contact lens should be removed before sleeping or swimming and should not be worn in the presence of noxious fumes or vapors. If unusual visual blurring occurs, the lens should not be worn until the condition subsides.

LENS CLEANING:
The RX-56® (porofocon A) contact lens should be cleaned daily with Preflex® solution, to remove mucous and film deposits.

LENS CARE:
The RX-56® (porofocon A) contact lens must be stored only in Flexsol® soaking solution. The lens should remain completely covered with this solution.

Before placing the RX-56® (porofocon A) contact lens on the eye, rinse the lens with Normol® solution. Discard the Flexsol® solution in the case, rinse the case with Normol® solution and leave case open to dry. Refill with fresh Flexsol® solution each time the lens is stored.

FITTING:
Conventional methods of fitting contact lenses can be used with the RX-56® (porofocon A) contact lens. For a detailed description of the suggested technique for fitting this lens, refer to the Professional Fitting Guide for the RX-56® (porofocon A) contact lens, copies of which are available from: Rynco Scientific Corporation, 31 Stewart Street, Post Office Box 270, Floral Park, New York 11002.

DOSAGE AND ADMINISTRATION:
STUDIES HAVE NOT BEEN PERFORMED TO SUPPORT SAFETY FOR WEAR OF THIS LENS BEYOND WAKING HOURS (EXTENDED WEAR).

WEARING SCHEDULE:
The following schedule suggests the proper wearing times for the RX-56® (porofocon A) contact lens:

Day	Hrs. Wear	Hrs. Rest	Hrs. Wear	Hrs. Rest	Hrs. Wear
1st	2	2	2	2	2
2nd	3	2	3	2	3
3rd	3	2	3	2	3
4th	4	1	4	1	4
5th	4	1	4	1	4
6th	5	1	5	1	Balance of waking hour
7th	5	1	5	1	Balance of waking hour
8th	6	1	6	1	Balance of waking hour
9th	6	1	6	1	Balance of waking hour
10th	7	1	7	1	Balance of waking hour
11th	7	1	7	1	Balance of waking hour
12th	8	1	8	1	Balance of waking hour
13th	8	1	8	1	Balance of waking hour
14th	All waking hours				

HOW SUPPLIED:
Each lens is supplied in a sealed sterile packet. The packet is labeled with the base curve, dioptric power, diameter, thickness and the lens identification number. When the lens is removed from its shipping case, it must be stored in Flexsol® solution for at least four hours prior to usage.

CAUTION:
Federal law prohibits dispensing without prescription

October 1978

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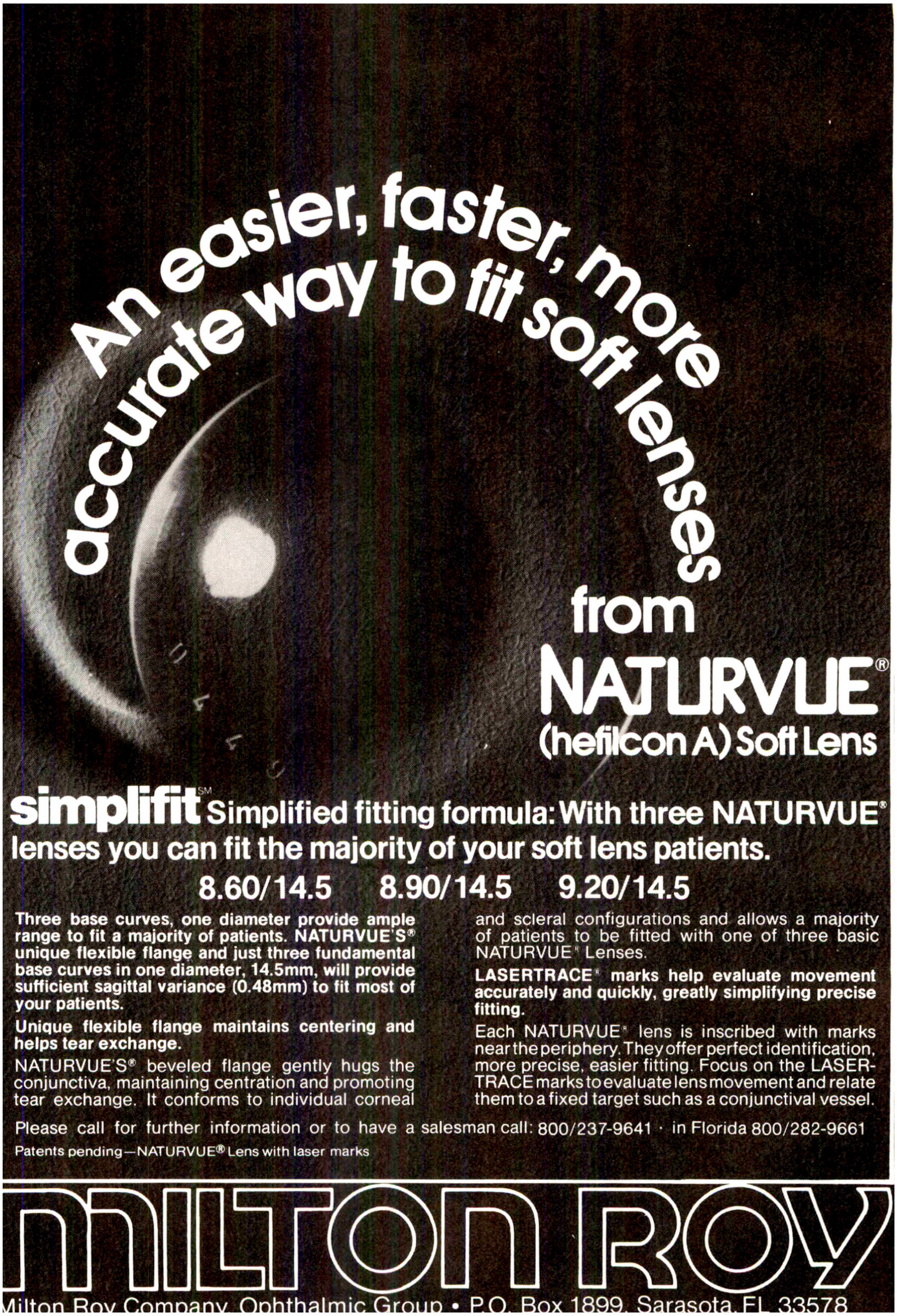
(1) Mandell, Robert B., Oxygen Permeability of Hard Contact Lenses. Contact Lens Forum, September 1977, pgs. 35-43.

*RX-56® is the Registered Trademark of RYNCO SCIENTIFIC CORPORATION. U.S. Patent #3,900,250.



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CONTRAINDICATIONS NATURVUE (hefilcon A) Contact Lenses are contraindicated by the presence of any of the following conditions:
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2. Any eye disease which affects the cornea or conjunctiva.
3. Insufficiency of lacrimal secretion.
4. Corneal hypoesthesia.
5. Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS The NATURVUE (hefilcon A) Contact Lens must be stored ONLY in BOILnSOAK® or FLEXSOL® solution, depending on the system for disinfection used. No ophthalmic solutions or medicants, including conventional hard contact lens solutions, can be used by NATURVUE (hefilcon A) Contact Lens wearers prior to, or while, the lens is in place on the eye. Only the recommended disinfecting soaking solutions BOILnSOAK or FLEXSOL, the recommended cleaner, PREFLEX®, or the recommended lubricating solution ADAPETTES® may be used on the NATURVUE (hefilcon A) Contact Lens.

Abrasions and Infections: If the lenses become less comfortable to the wearer than when they were first placed on the wearer's corneas, this may indicate the presence of a foreign body. The lenses should be removed immediately and the patient examined. If any scratching, redness or pain is present, a physician should be consulted immediately.

Lens Care: Patients must adhere to recommended daily care of the NATURVUE (hefilcon A) Contact Lenses. Failure to follow this procedure may result in development of serious ocular infections.

Wearing Restrictions: The NATURVUE (hefilcon A) Contact Lens should be removed before sleeping or swimming or in the presence of noxious and irritating vapors.

Visual Blurring: When visual blurring occurs the lens must be removed until the condition subsides.

PRECAUTIONS: Storage– NATURVUE (hefilcon A) Contact Lenses must be stored ONLY in BOILnSOAK or FLEXSOL solution. If left exposed to air, the lenses will dehydrate. If a lens dehydrates, it should be soaked in BOILnSOAK or FLEXSOL solution until it returns to a soft, supple state.

CLEANING AND DISINFECTING: NATURVUE (hefilcon A) Contact Lenses must be both cleaned and disinfected daily.

Thermal Disinfection: Cleaning with PREFLEX solution followed by disinfection with the NATURVUE Thermal Unit and BOILnSOAK has been shown to be an effective system for daily care of the lens. If a Thermal Unit is temporarily not available the lenses may be boiled in the carrying case filled with BOILnSOAK in a pan of water for 10 minutes.

Chemical Disinfection: Cleaning with PREFLEX, disinfection with FLEXSOL and rinsing with NORMOL® solutions has been shown to be an effective system for the daily care of the lenses.

WARNING DO NOT MIX OR ALTERNATE THE DISINFECTION AND STORAGE SYSTEMS, FLEXSOL SHOULD NOT BE USED WITH HEAT.

Patient Care: Patients must be instructed to wash and rinse hands thoroughly and dry with a lint-free towel before handling the lenses.

Cosmetics, lotions, soaps and creams must not come in contact with the lenses because eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until hair spray has settled.

Fluorescein: Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with normal saline solution and wait at least one hour before replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescein irreversibly.

ADVERSE REACTIONS Serious corneal damage may result from wearing a lens which has been soaked in a conventional hard contact lens solution.

Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritations.

On rare occasions a lens may adhere to an eye as a result of the patient sleeping with the lens on or wearing a hypotonic lens. If a lens adheres for any reason, patients may be instructed to apply a few drops of BOILnSOAK or ADAPETTES solution and wait until the lens moves freely before removing it.

Rainbows or halos around objects or blurring of the vision may occur if the lenses are worn continuously for too long a time. Removal of the lenses and a rest period of at least one hour generally relieves these symptoms.

Excessive tearing, unusual eye secretions, and photophobia are not normal; if these occur, the patient should be examined by a practitioner to determine their cause.

DOSAGE AND ADMINISTRATION For a description of the fitting technique, refer to the NATURVUE Fitting Guide available from Milton Roy Company, P.O. Box 1899, Sarasota, FL 33578.

It is important for the patient to adhere to the daily wearing schedule shown in the Patient Instruction Manual.

In Use Durability: With normal use and care, NATURVUE (hefilcon A) Contact Lenses have been shown to be durable for a minimum of 1 year.

HOW SUPPLIED Each lens is supplied sterile in a glass vial containing sterile saline solution. The vial is marked with the lens parameters and the manufacturing lot number. To assure proper lens care and handling, each NATURVUE patient must be supplied with a complete NATURVUE Patient Care Kit.

For full disclosure of prescribing information, consult the NATURVUE Instruction Manual. Caution: Federal law prohibits dispensing without prescription. Patents Pending—NATURVUE lens with laser marks.

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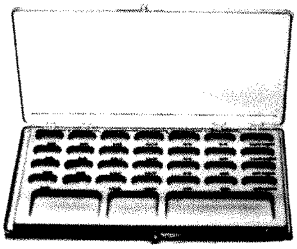

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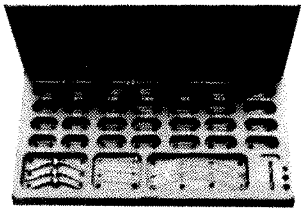

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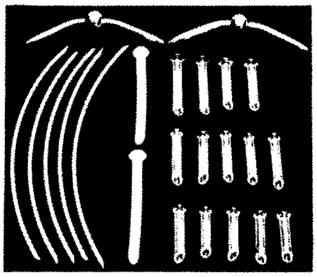

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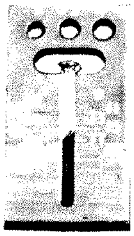

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* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
J.C. Mustarde', L.T. Jones M.D. and A. Callahan M.D., Ophthalmic Plastic Surgery—Up-To-Date, Aesculapius Publishing Company, 1970.
L.T. Jones M.D. and J.L. Wobig M.D., Surgery of the Eyelids and Lacrimal System, Aesculapius Publishing Company, 1976.

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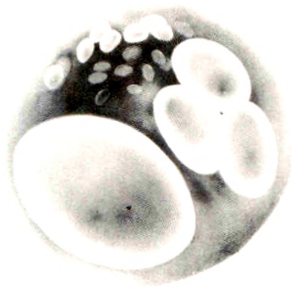

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REFERENCE: [1]Antibacterial Agents. Monograph I. *Office Seminars in Ophthalmology*." Irving H. Leopold, ed. Allergan Pharmaceuticals, 1976, p. 5.

ALLERGAN Pharmaceuticals, Inc.
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AMERICAN JOURNAL OF OPHTHALMOLOGY

VOLUME 87 NUMBER 6 JUNE, 1979

AN EVALUATION OF ENUCLEATION IN THE MANAGEMENT OF UVEAL MELANOMAS

LORENZ E. ZIMMERMAN, M.D., AND IAN W. MCLEAN, M.D.
Washington, D.C.

Enucleation has long been the standard method for treating uveal melanomas and retinoblastoma. Although the course of untreated retinoblastomas and the life-saving importance of early diagnosis and prompt treatment of these tumors have been well documented, the same is not true of uveal melanomas. The rationale for enucleation of uveal tumors has been based more on assumptions than on a firm foundation of data derived from good clinical and pathologic studies of untreated tumors. The principal assumptions are that malignant melanomas of the uvea, like those of the skin, have the potential to produce metastatic disease early in their course, and that early diagnosis and prompt enucleation are necessary if one is to prevent metastasis and early death.

Although an enormous number of published reports of the past century have dealt with various aspects of uveal melanomas, we still have almost no information as to the natural course of untreated tumors. How often and how rapidly do discoid tumors measuring no more than 10 mm in diameter or 2 mm in elevation progress to tumors exceeding 15 mm in diameter or 5 mm in elevation? How long does it take for small tumors to grow large enough to cause blindness from retinal detachment, opacification of the media, secondary glaucoma, and the like? How often and at what stage in the natural course of untreated tumors will metastatic disease be encountered? One would think that the previously published material provided substantial information bearing on these questions, and that enucleation was adopted as the standard treatment because of information derived from untreated patients, but such does not seem to have been the case. In 1882, Fuchs[1] indicated that all intraocular melanomas were treated by enucleation and that untreated cases were reported only in the "older literature." He cited three untreated cases in which death followed a clinical course that lasted 5½, 3¾, and 1⅔ years, respectively. More than 20 years later, Parsons[2] cited only the same three cases.

From the Department of Ophthalmic Pathology and the Registry of Ophthalmic Pathology, Armed Forces Institute of Pathology, Washington, D.C. This study was supported in part by Research Grant EY-01386 from the National Eye Institute, National Institutes of Health, Bethesda, Maryland.

Portions of this paper were presented by Dr. Zimmerman as the Sixth C. Dwight Townes Memorial Lecture, University of Louisville, Louisville, Kentucky, March 10, 1978, and formed the basis of the Donders Medal Lecture given before the Netherlands Ophthalmological Society in Groningen, The Netherlands, March 30, 1978. Much of this material was also presented by Dr. Zimmerman in the Estelle Doheny Memorial Lecture given in Los Angeles, Dec. 15, 1978.

The opinions and assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

Reprint requests to Lorenz E. Zimmerman, M.D., Armed Forces Institute of Pathology, Washington, DC 20306.

Fuchs[1,2] is often quoted as having had a cure rate of only 6%, but this should not be interpreted as evidence of 94% fatality. Fuchs collected 259 cases of which only 23 were his own. His criterion for a "definite cure" was known survival for four years or more after enucleation with no evidence of local recurrence or of metastatic disease. Only 16 (6%) of these cases met this criterion. Conversely, he described metastasis in only 48 (18.5%) cases. We must assume that he had inadequate information in 195 (75%) of his collected cases. If one considers solely the 16 patients believed to have been cured and the 48 known to have developed metastasis, then the cure rate was about 25% as of 1882. In 1891, Lawford and Collins[3] reviewed the cases of 79 patients followed up at Moorfields Hospital and found a three-year recovery rate of 25%. These authors[3] cited somewhat higher recovery rates reported by Hirschberg (38%) and Freudenthal (37.5%).

Before the turn of the century intraocular melanomas were often far advanced by the time they were discovered and treated. Thus, Lawford and Collins described[3] extraocular extension clinically evident in 20% of the cases and optic nerve invasion in 22%. Two thirds of the eyes were glaucomatous. Yet, despite such advanced neoplastic disease, no instance of preoperative metastasis was described and the earliest postoperative death occurred after six months.

The rationale for enucleation as the treatment for uveal melanomas at the turn of the century cannot be disputed: (1) most tumors were advanced when they were discovered; (2) the eye was always symptomatic and often blind from complications produced by the tumors; (3) at least three deaths from untreated tumors were on record; and (4) no alternate methods of treatment were available. That all melanocytic tumors were not equally malignant was unknown for many years. Nor was it known that uveal melanomas were generally less malignant than most melanomas of the skin. Additionally, the concept of surgical excision of every localized malignant neoplasm had become widely established.

von Hippel[4,5] evaluated the results of treatment of uveal melanomas. He concluded that despite many publications concerning the topic, it was not possible to document the need to enucleate an eye immediately after the diagnosis of an intraocular tumor. He found little evidence that early enucleation saved more lives than if enucleation were delayed until the eye became glaucomatous. In his own series he had a lower incidence of metastatic disease (34%) in stage I (nonglaucomatous) than in stage II (glaucomatous) (47%), but some very small tumors produced metastatic disease and death. von Hippel supported Lange's belief that the body's immunologic defense mechanisms were probably of paramount importance in determining outcome, which necessitated long-term follow-up studies, which were notably lacking at the time.

During the past 30 years increasing numbers of uveal melanomas have been detected while they were still small, and often asymptomatic, whereas a decreasing proportion were as far advanced as the typical cases in the series of Lawford and Collins.[3] During this same period, it has become widely recognized that most melanomas that measure no more than 10 mm in diameter or 3 mm in thickness, including almost all iridic tumors, have had a benign course after enucleation.[6-10] Most are composed of the less highly malignant spindle-shaped melanoma cells, and some have been shown to be cytologically benign nevi.[11-13] Consequently, more effort has been made to salvage tumor-containing eyes with useful vision by development of new techniques for surgical resection (iridectomy, iridocyclectomy, goniectomy, full-thickness posterior eyewall resection, and the like); for radiation therapy (cobalt plaques, radon seeds, proton beams, and the like); or thermal coagulation (photocoagulation, cryotherapy, and the like). As these newer methods of treatment are introduced and sometimes used in various combinations, evaluation of results becomes increasingly more difficult because of the lack of information regarding the natural course of untreated tumors. Results of treatment by these other methods, however, are not our main concern in this article. Our focus centers on results obtained by enucleation. After enucleation, the only significant criteria are whether the patient survives or dies as a consequence of metastatic disease, and whether the operation gives the patient a better chance to achieve normal life expectancy. Jensen[14] and Vogel[15] summarized much of the follow-up data. They found five-year survival rates ranging from 14 to 84%, but no particular trend was evident. For example, one of the highest survival rates reported was from a series of 250 patients treated at two ophthalmic hospitals in London before 1940.[16] Since then almost all authors have recorded much lower five-year and ten-year survival rates. In 1972, Vogel[15] wrote:

> The published mortality rates following such enucleation indicate that the results are either disappointing or at least quite controversial.

In 1977 Curtin[17] stated:

> The overall death rate of 50 percent in ten years in patients treated by enucleation at the time of diagnosis does little to inspire confidence in our present management of the disease.

More than 20 years ago, Westerveld-Brandon and Zeeman[18] used a new approach to evaluate results obtained by enucleation. Instead of ascertaining five-, ten-, or 15-year mortality, they set out to determine how many patients reached normal life expectancy. They arranged their collected data according to the age at onset of symptoms, age at time of enucleation, and age at death or last examination. They evaluated these data with respect to normal life expectancy. They concluded:

> The theory that immediate destruction of the neoplasm or enucleation is indicated in cases of choroid melanoblastoma in order to prolong life is not supported by the above-mentioned facts; enucleation of an eye which is still functionally sound on the basis of this theory would, therefore, seem to be unjustifiable.

During recent years, interesting follow-up information has been presented in the form of actuarial survival curves, which have been useful in vividly contrasting the prognostic significance of such variables as cell types, size of tumors, age of patients, and the like.

Several problems are inherent with the data on which these actuarial curves are based: (1) The cause of death is more often determined from death certificates than from postmortem studies; (2) the actuarial survival curves have rarely been compared with those of matched populations of nonmelanoma patients of similar age distribution; (3) the mortality before treatment or among nontreated patients is not known. For example, in the survival curves shown in Figure 1 from the studies of Paul, Parnell, and Fraker,[19] the most striking differences in the slopes of

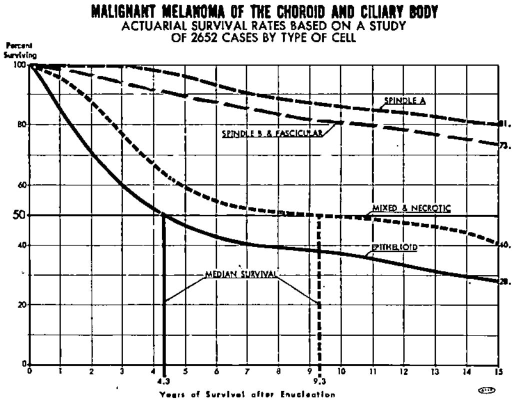

Fig. 1 (Zimmerman and McLean). Actuarial curves published by Paul, Parnell, and Fraker[19] showing the relationship of survival to cell type in a series of 2,652 patients who had been treated by enucleation of the melanoma-containing eye. The curves for melanomas of epithelioid and mixed cell type have a steep downward slope beginning soon after enucleation.

these curves occur during the first several years after enucleation. Thereafter, the slopes of the curves become similar. Perhaps the tumor deaths during the later years of this study did not include many deaths from new unrecognized primary neoplasms. The steep downward slope of the curves for patients whose tumors were of the epithelioid or mixed cell types suggests that these are tumors in which there should be an appreciable rate of mortality before enucleation. However, evidence of metastatic disease before or at the time of discovery of the intraocular tumor has rarely been documented.[20] Of even greater clinical significance is that little documentation is available to support the concern (which is admittedly less prevalent today than it was 20 years ago, though still evident from the cases we see, the communications we receive, and some of the current published material[21]) that a delay in treating a small tumor may significantly jeopardize the patient's chances of surviving.

We believe the following points must be more emphasized and better documented.

1. Uveal melanomas, unlike those of the skin, have no potential for early production of metastatic disease.* Evidence that uveal melanomas are generally slow-growing tumors is increasing. Part of the evidence is derived from a retrospective analysis of patients' symptoms and from cases in which the patient has been treated for uveitis, glaucoma, or retinal detachment before a tumor was suspected. Other evidence is based on clinical studies of untreated tumors that have shown minimal change or slow growth over a period of years. Dunphy[22,23] twice described one of his patients who refused the recommended enucleation until the eye had become stony hard, blind, and painful 14 years later. Pathologic study of the enucleated eye established that "the three ophthalmologists had been quite right in their diagnosis but the only trouble was that they were all now dead, whereas the patient was in excellent health." This was a melanoma of the iris and ciliary body. Today it is generally known that melanomas originating in the iris have a more favorable prognosis than those originating in the choroid.

At the 1977 meeting of the Verhoeff Society, Albert[24] described another patient whose tumor-containing eye had been examined by five ophthalmologists, all of whom concurred in the diagnosis of malignant melanoma of the choroid. Because the eye retained good vision, the patient refused enucleation. She was examined at intervals of about six months for a period of more than 25 years after which time she finally noted a marked reduction in vision. A retinal detachment had developed and her visual acuity was 6/60 (20/200). The patient then agreed to enucleation. Histopathologic examination revealed the melanoma to be of mixed cell type. Although no evidence of metastatic disease had been observed during the exceptionally long period of preoperative clinical observation, Daniel Albert (personal communication, January 1978) informed us that the patient died of metastatic disease during the second postoperative year.

Koenig[25] described a case documenting the slow growth of intraocular melanomas for periods of more than 20 years without producing metastatic disease. The other eye had been enucleated (for unstated reasons) three years earlier by another ophthalmologist, so the patient and her family would not permit enucleation of the tumor-containing eye. Koenig was, therefore, able to observe the slowly enlarging tumor for 20 years until the patient died as a result of a coronary occlusion. The eye was obtained after death and the intraocular melanoma was found to be of the mixed cell type; there was no evidence of metastasis.

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*Although current concepts suggest that uveal melanomas, like many other malignant tumors, may release malignant cells into the circulation over extended periods of time, such cells are usually eliminated by an effective immunologic defense mechanism before metastatic foci develop. Thus, to suggest that uveal melanomas may disseminate cells early, yet rarely produce metastatic disease until late, is consistent.

Metastatic spread of uveal melanoma before the primary tumor has become symptomatic is almost unknown. Most of the small, asymptomatic, and untreated tumors observed by Char and Hogan,[26] Curtin,[17] Gass,[11] Hagler, Jarrett, and Killian,[27] Shields,[28,29] and others have shown little or no growth and no evidence of metastasis.

There are exceptions to the axiom that untreated uveal melanomas rarely metastasize before growing larger and causing intraocular complications. Ruiz[21] recorded the case of a woman who had a small choroidal melanoma that resulted in widespread metastasis nine years after it was first observed, but before it had shown any signs of local growth or retinal detachment.

Although most ophthalmologists and ophthalmic pathologists rarely see a patient with an untreated uveal melanoma with metastasis, quantitative data are not available. Having polled a number of clinicians who have seen an aggregate of several thousand patients with uveal melanomas, we estimate that not more than about 1% of the patients show evidence of metastatic disease before enucleation.

Jensen[14,30] analyzed all cases of malignant intraocular melanomas, recognized as a result of clinical or histopathologic studies, or both, throughout Denmark during 1943-1952. There were 295 malignant melanomas of the choroid and ciliary body and ten of the iris. In no case was metastasis discovered before enucleation. Furthermore, although evidence of metastatic disease was seldom observed during the first six months after enucleation, it became evident in most fatal cases during the second or third years; 85% of the patients died within the ensuing six months. During this ten-year period there were no totally untreated tumors, but enucleation was delayed in 12 cases for periods ranging from one to 5 1/2 years. There was no evidence that the delay in enucleation resulted in an increased frequency of metastatic disease. Jensen subsequently found two cases of untreated uveal melanoma at autopsy. The patients were 85 and 90 years old, respectively. These two cases were included in a series of 29 case reports of untreated uveal melanomas causing metastasis.[20] Fifteen of the 29 cases in that series were drawn from a total of 3,073 uveal melanoma cases on file in the Registry of Ophthalmic Pathology, an incidence of 0.5%.

Raivio[31] reported that there were 32 of 378 cases of uveal melanoma in Finland in which the tumor-containing eye had been observed for some time before enucleation. Although there was growth in each instance, no metastasis was observed and the postoperative survival was no worse than among those whose eyes had been enucleated promptly after diagnosis. Five additional patients refused enucleation. One died of unrelated causes. The other four eventually died of metastatic disease but in none did the tumor prove lethal in less than two years, the deaths occurring after two, three, 14, and 15 years, respectively.

Sobânski and associates[32-34] described ten patients with uveal melanomas who refused enucleation; all died of metastatic disease within ten years. Sobânski provided us with additional information (personal communication, May 1978). In four cases the tumors occupied about one-fourth of the globe and produced little clinical disturbance. These four patients survived nine, four, two, and two years, respectively, after they had refused enucleation. In six cases the tumors filled the globe, causing glaucoma and blindness. Patients survived eight, five, two, two, one, and one years, respectively.

In the combined series of Raivio[31] and Sobânski and associates[32-34] there were 14 untreated patients who died of metastatic disease. The average survival was five years without treatment; six of the patients lived four to 15 years after refusing enucleation.

Morgan[35] stated that he has "known simple-looking spindle cell tumours of the choroid, in the absence of orbital spread, to metastasise early." Upon inquiry, we learned that he meant those cases in which metastasis had been discovered within a year of enucleation. He had seen no cases in which metastatic disease had developed before enucleation and knew of no such cases at the Institute of Ophthalmology in London.

Char[36] and Einhorn, and associates[37] described the metastatic patterns of choroidal melanomas from two large series and had no cases in which metastasis was the result of an untreated tumor. More recently, Young[20] reported that eight of 122 patients admitted to the M. D. Anderson Hospital in Houston, Texas, for treatment of metastatic disease from uveal melanomas had either had no treatment of the primary tumor, or metastatic disease was evident at the time of enucleation. One was the case of a young woman previously recorded by Ruiz.[21] By striking contrast, the other seven patients were all much older with advanced growth of the primary tumors.

Reese and Howard[38] reported on a series of 38 cases of diffuse, malignant melanomas; Font, Spaulding, and Zimmerman[39] recorded data on 54 cases. Many patients in both series had prolonged treatment for glaucoma, uveitis, or retinal detachment, and an intraocular tumor was not suspected. Both reports emphasized the long duration of ocular symptoms before enucleation, which ranged up to seven years in one series and up to nine years in the other. Extraocular extension, often multiple, was frequent and the tumor cells became disseminated throughout the orbit, optic nerve, and subarachnoid space. Despite the advanced state of the melanomas and the frequency of extraocular extension, there were no cases in either series in which preoperative metastasis was found. Only three of the 19 tumor deaths in one series[39] occurred during the first year after enucleation. The peak mortality was during the second year. Although Reese and Howard[38] did not provide postoperative follow-up data, none of their patients showed evidence of metastasis before enucleation. Spencer and Iverson[40] described a man who developed heterochromia and cyclitis 12 years earlier and had been treated for an ipsilateral glaucoma nine years earlier. After the eye had been blind for two years it was enucleated. Histopathologic examination of the eye revealed widespread intraocular dispersion of melanoma cells derived from a diffuse melanoma of the iris with massive invasion of the optic nerve. Despite the long history and optic nerve invasion, there was no clinical evidence of metastatic disease until 22 months after enucleation (personal communication from W. E. Spencer, May 1978).

Eyes have been obtained after death as part of the complete autopsy at the Johns Hopkins Hospital for many years, but in the entire history of that institution there has been only one death from metastasis of an untreated uveal melanoma (personal communication from W. Richard Green, June 1978). There were 57 melanoma deaths of which eight were attributed to intraocular sites of origin but in seven of the eight cases the tumor-containing eye had been enucleated before metastatic disease developed. In his postmortem studies, Dr. Green has also found two unsuspected intraocular melanomas that had not metastasized; these were incidental findings in patients who had died of unrelated diseases (personal communication, June 1978). Although for many years eyes have been removed after death at Barnes Hospital in St. Louis, Morton Smith stated that he has not been able to identify any case in which metastasis was attributable to an untreated intraocular melanoma (personal communication, February 1978). In a study of the eyes obtained at autopsy in 4,477 deaths at the University of California, Los Angeles, Robert Foos found no fatalities attributable to untreated intraocular melanomas (personal communication, April 1978).

Thus, the clinical observations of the most experienced ophthalmologists, the reports describing untreated uveal melanomas, the long period of ocular symptoms with flat diffuse uveal melanomas, and the postmortem data support our conviction that uveal melanomas do not metastasize early.

2. The dictum, "early recognition and prompt enucleation," is an unwarranted radical approach to solving the melanoma problem. Many authors who advocate early enucleation for melanomas cite our own reports[6,8,9] and those of other investigators[7,10] to establish that the prognosis for life after enucleation is much worse for those patients with large tumors than for patients with small tumors. Shammas and Blodi[10] compared actuarial survival curves for patients with tumors larger and smaller than 10 mm and showed a striking difference. Patients whose tumors measured less than 10 mm had a normal life expectancy, whereas those whose tumors were larger had a significant early mortality after enucleation.[10] Although such data may be used to justify early enucleation when tumors measure less than 10 mm, these same data can be used to support the contention that most small tumors differ biologically and in their natural course are essentially benign.

3. Despite the advances that have been made in clinical diagnosis and the availability of such specialized diagnostic methods as fluorescein angiography, ultrasonography, and the phosphorus uptake (^{32}P) test, correct preoperative diagnosis of small lesions continues to pose a serious problem. Although Ferry[41] and Shields and Zimmerman[42] recorded a high incidence (19 to 20%) of false-positive diagnosis of uveal melanoma even though the lesion in question could be examined through clear media, more recent writers have claimed much improved results.[43-48] Nevertheless, there is a continuing problem in distinguishing between large nevi and small melanomas.[11,49-55] All of the clinical signs and specialized tests that have been used to differentiate nevi from melanomas have exceptions. Nevi may grow,[11-12] cause serous or hemorrhagic detachments of retina or retinal pigment epithelium,[11] produce field defects,[53,55] show early fluorescence and late leakage on angiography, extend through the sclera,[51] and retain ^{32}P at abnormally high levels.[12,13] They tend to remain flat and rarely become thicker than 1 mm (corresponding to 3 diopters of elevation),[11,53,54,56] but we have seen one choroidal nevus (melanocytoma type) that infiltrated the retina through a rupture of Bruch's membrane.

There is no possibility for histopathologic confirmation of the diagnosis when small tumors suspected of being malignant melanomas are not treated by excision or enucleation. Even with histopathologic "documentation," we believe that Gass[11] was correct when he suggested that pathologists overdiagnose malignant melanoma. Thus, we found in a recently completed critical reevaluation of spindle A melanomas on file in the Registry of Ophthalmic Pathology, that in 15 (14%) of 105 cases there was neither convincing clinical nor cytopathologic evidence that the tumors were malignant.[12] These tumors have now been redesignated as spindle-cell nevi. This failure to separate spindle-cell nevi from spindle cell malignant melanomas is partly responsible for many clinicians' belief that disproportionately large visual field defects, early fluorescence and late leakage on angiography, and a positive ^{32}P test reliably differentiate choroidal malignant melanoma from choroidal nevi.

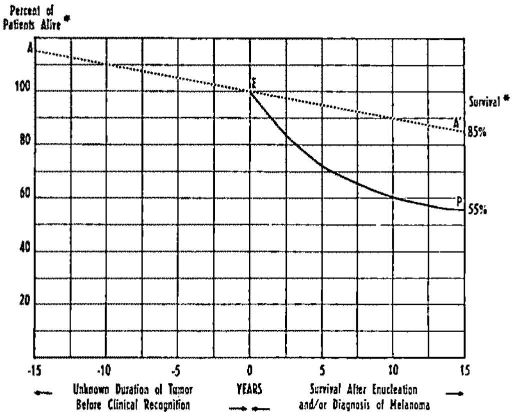
PROGNOSIS

The many writings concerning prognosis and prognostic factors do not adequately emphasize that nearly all such data are based on clinicopathologic studies of the tumor-containing eye after enucleation. Thus, conclusions that melanomas containing epithelioid cells, tumors of large size, and those with greater mitotic activity, extraocular extension, or vascular invasion are the ones with a high risk of producing a lethal outcome, are based on cases in which the natural course of the tumor was interrupted by enucleation. Few conclusions concerning prognosis can be based on data derived from untreated melanomas. Thus, the expression "natural history of malignant melanomas" has been improperly used by some investigators,[7,35] who made no observations on untreated patients.

One of the best ways of comparing prognostic factors is by means of actuarial curves.[6,10,14,19] Such curves, however, may be misleading in suggesting that the biologic experiment began at time zero.[57] Actually, however, time zero is the time when the tumor-containing eye was enucleated. In most cases this was also the time when the presence of the intraocular melanoma was discovered because in the past there seldom has been any great interval between discovery of the tumor and enucleation.

If removal of the tumor-containing eye had a beneficial effect, this could be demonstrated by comparing the survival curves of postenucleation patients with those of untreated patients with intraocular melanomas. The survival curve of the untreated would have a much steeper downward slope, indicative of a worse prognosis.

Unfortunately no reliable data exist concerning untreated melanomas and we can only guess as to the frequency with which uveal melanomas cause metastatic disease or death, or both, before treatment. We have estimated the death rate from untreated uveal melanomas to be about 1% per year.[57] Using this estimate to indicate the hypothetical survival curve of untreated patients with uveal melanomas, as compared to the survival curves for patients treated by enucleation (Fig. 2), we conclude that this operation must have an adverse rather than a beneficial effect. Thus, "poor prognosis factors" may indicate that large tumors containing epithelioid cells have a much greater chance of widely disseminating tumor cells than have small tumors composed of cohesive spindle cells. Large tumors containing poorly cohesive epi-

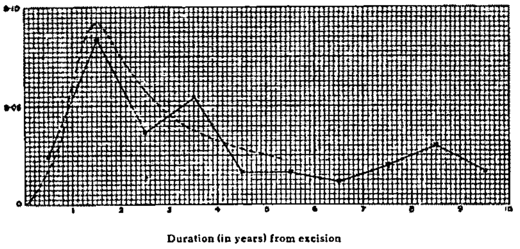


*Calibrated so that 100% ≈ number patients alive at time of discovery and/or enucleation (E).

Fig. 2 (Zimmerman and McLean). Actual and hypothetical survival curves for patients with malignant melanoma of the choroid and ciliary body. The dotted line A-A′ is a hypothetical survival curve of untreated patients assuming an annual mortality of 1% per year. E represents that point in time when the natural course of the tumor is interrupted by enucleation of the eye. E-P is the actuarial survival curve based on the follow-up data on the 2,652 cases in the series of Paul, Parnell, and Fraker.[19] The steep downward slope of curve E-P during the first five years after enucleation as compared with the expected survival curve E-A′ suggests that enucleation has a detrimental effect.

thelioid cells are also much more likely to have large thin-walled vascular channels and foci of intravascular extension of tumor cells.

The changing risk of death from metastatic disease after enucleation is calculated from the same data used to draw survival curves after enucleation. If all cases are pooled, about six months after enucleating the risk of a fatal outcome begins to rise, from the preoperative low of about 1% per year, to a peak of about 8% per year during the second postoperative year, and then falls slowly during the next few years to reach the original level of about 1% per year seven to eight years after enucleation (Fig. 3).[57] About two thirds of these tumor deaths may be attributed to the adverse effects of enucleation. Benjamin and associates[16] published a strikingly similar curve for annual mortality peaking at 8% during the second year after enucleation (Fig. 4), but they made no comment about its possible significance. From Jensen's data[14,30] (and personal communication, 1978) we have plotted annual mortality and again found a sharp peak (12% per year) during the second postoperative year (Fig. 5).



Central death ratio (*m*). (Ratio of deaths from cancer to mean lives exposed during the year). The discontinuous line indicates the underlying trend. In view of the restricted data no attempt was made to fit a curve of a specific mathematical law but an approximation thereto was obtained by drawing a curve through the observed values by graphic graduation.

Fig. 4 (Zimmerman and McLean). Relationship of annual death rate to time after enucleation in the series reported by Benjamin and associates,[16] who assumed a mortality of zero before enucleation. The broken line represents a visual approximation of the actual data points. Observe the similarity of this curve to that shown in Figure 3. (Reprinted with permission of the authors and the editor, British Journal of Ophthalmology.)

By contrasting the survival data for

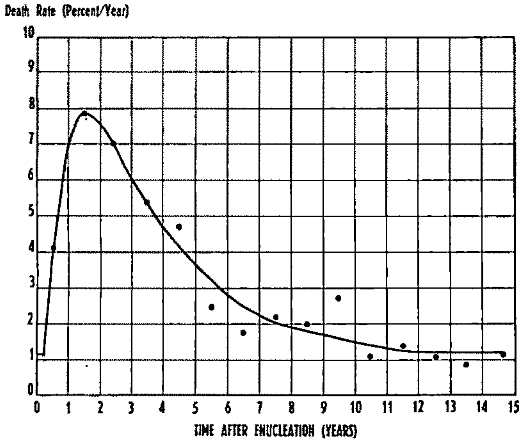


Fig. 3 (Zimmerman and McLean). Relationship of annual death rate to time after enucleation. After a lag period of several months the death rate rises abruptly, reaching a peak of about 8% per year during the second year after enucleation. It then falls off over a period of several years, reaching the estimated pre-enucleation rate of about 1% per year. The smooth curve represents the least-square fit to the observed death rates using the model described by Zimmerman, McLean, and Foster.[57] (Reprinted with the permission of the authors and the editor, British Journal of Ophthalmology.)

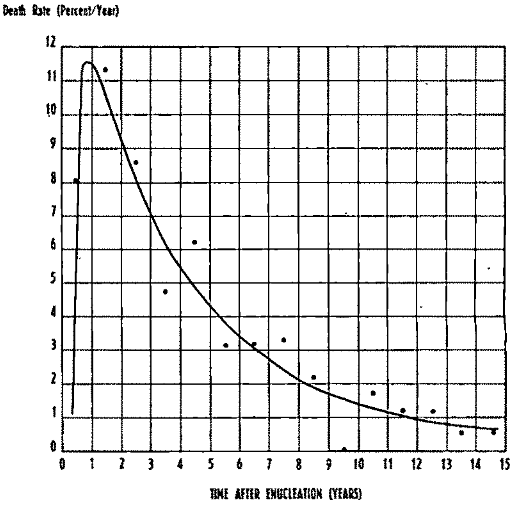


Fig. 5 (Zimmerman and McLean). Relationship of annual death rate to time after enucleation based on data provided by Jensen from his series.[14,30] The smooth curve represents the least-square fit to the observed death rates, using the equation formulated by Zimmerman, McLean, and Foster.[57] Although the peak during the second year is higher, this curve is similar to those shown in Figures 3 and 4.

pure spindle-cell melanomas and tumors containing epithelioid cells, or comparing large vs small melanomas, instead of pooling all cases, the peak annual death rate is much higher for patients with large tumors, or tumors containing epithelioid cells, or both, as compared with that for those whose tumors were small or composed only of spindle cells, or both.

THE NATURE OF UVEAL MELANOMAS

With respect to the natural biologic potential of uveal melanomas, great differences in behavior exhibited by various cancers must be considered. Some, such as basal cell carcinomas of the inner canthus, gliomas of the brain, and retroperitoneal liposarcomas, are typically locally aggressive neoplasms that may prove lethal, even though they rarely metastasize. At the opposite end of the spectrum are such neoplasms as the "oat cell" bronchogenic carcinoma and certain carcinomas originating in the stomach, pancreas, and prostate that frequently metastasize before the primary site is discovered. Still another phenomenon exhibited by some tumors (for example, chondrosarcomas of the pelvis, hepatomas, and certain renal cell carcinomas) is a special form of vascular invasion in which the neoplastic cells grow in a compact cohesive manner within the lumina of major vessels draining the tumor without becoming disseminated. We have studied cases in which tumor spread up the vena cava and finally killed the patient by almost total obstruction of all venous return to the right auricle. Yet there was no disseminated metastatic disease. As to where in this spectrum of neoplastic potential uveal melanomas belong, we believe evidence indicates that uveal melanomas belong with those cancers that characteristically exhibit slow but progressive local invasiveness, usually without the production of metastatic disease until late in their natural course. Many also show a striking tendency to invade major blood vessels, even filling a vortex vein without necessarily resulting in metastasis.

PATHOGENESIS OF METASTATIC DISEASE

The increased mortality from metastatic disease that follows removal of a melanoma-containing eye may occur because of manipulations of the globe, extraocular muscles, orbital tissues, and optic nerve; and the attendant large fluctuations in intraocular pressure during the surgical procedure may greatly increase the intravasation and dissemination of tumor cells.[57-60] Additionally, the removal of the tumor may adversely affect the host's immunologic defense mechanisms,[61-65] or for other unknown reasons, may correlate with a breakdown of such defenses.[8,57]

Fraunfelder and associates,[58,59] Wilson and Fraunfelder,[60] and others who have measured intraocular pressure during enucleation have shown that isolation of the rectus muscles, passage of the muscle hook under the muscle, lifting the muscle to allow insertion of the scissors before cutting, placement of the enucleation scissors, grasping of the optic nerve, manipulations of orbital fat, scleral depression as required for various diagnostic procedures, application for ^{32}P test probes, ocular massage, and even the forceful closure of the eyelids may produce increases in intraocular pressure up to 500 mm Hg. Burton[66] described rupture of Bruch's membrane and intraocular hemorrhage during such diagnostic procedures as transillumination and ^{32}P testing.

The numerous dilated, thin-walled blood vessels and blood lakes present in many uveal melanomas[2,67-72] assume greater significance in view of the possible effects of surgical trauma. It seems only logical that any great fluctuations of intraocular pressure might create significant hydrodynamic disturbances within a

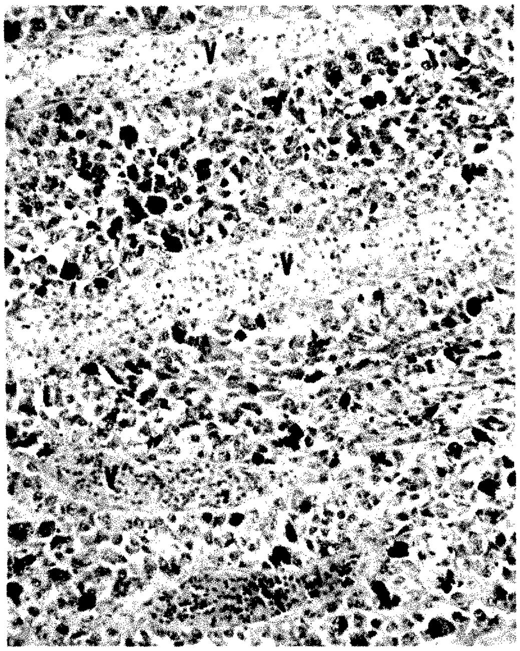


Fig. 6 (Zimmerman and McLean). Many widely dilated thin-walled vascular channels (V) course through this malignant melanoma, which is predominantly composed of poorly cohesive epithelioid cells (×160).

large highly vascular melanoma containing poorly cohesive epithelioid cells (Fig. 6), leading to a disintegration of the delicate layer of endothelial cells that separate tumor cells from the circulating blood. Microscopic examination of uveal melanomas often shows hemorrhages and blood lakes that may have developed during enucleation. With a breakdown in the integrity of the vessel wall, additional fluctuations in intraocular pressure may facilitate the intravasation of neoplastic cells as well as the extravasation of blood into the tumor.

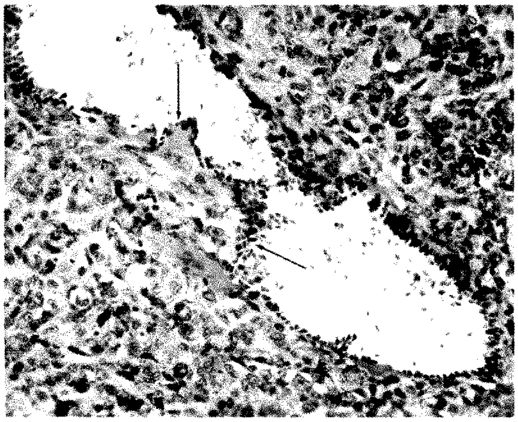


Fig. 7 (Zimmerman and McLean). Segment of a large thin-walled vein within a choroidal melanoma containing a focus in which the tumor cells have penetrated into the lumen (arrows) (×250).

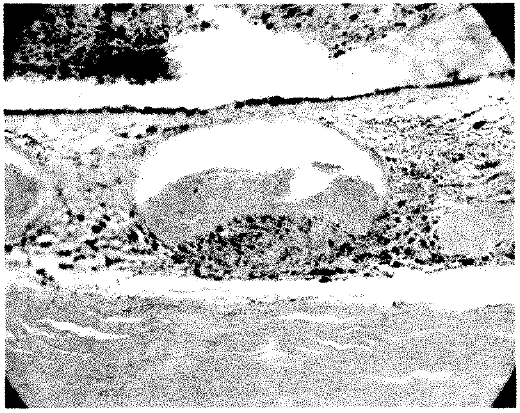


Fig. 8 (Zimmerman and McLean). A plaque of tumor cells occupies part of the lumen of a large tributary of a vortex vein at the periphery of a large melanoma of the choroid (×125).

Uveal melanomas also exhibit foci in which tumor cells have invaded vessel walls (Fig. 7), or tumor growth may be observed along the luminal surface of vessels within the tumor (Fig. 8), in

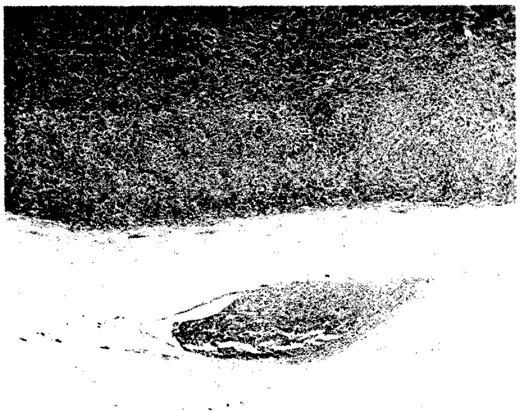


Fig. 9 (Zimmerman and McLean). Much of the lumen of this vortex vein within a scleral canal is filled with tumor (×50).

scleral canals (Fig. 9), or in a vortex vein on the episcleral surface (Fig. 10). Cells from such intraluminal growths may become dislodged as a consequence of the many great fluctuations of intraocular pressure that take place during enucleation. Histologic observations document that large masses of tumor tissue may become dislodged into the circulation (Fig. 11). Efforts to document intravasation of tumor cells in vivo by cytologic studies of blood have had inconclusive results.[73,74]

Histopathologic studies support the thesis that enucleation is less likely to disseminate cells if the tumor is small, of the spindle cell type, or both. Large tumors and those containing many epithelioid cells are more likely to have large thin-walled vascular channels with or without intraluminal extension of the neoplasm than are small tumors and those composed entirely of tightly cohesive spindle cells, which are also much less vascular. Small tumors are less vulnerable to dissemination because of the cohesive character of the constituent cells. This cohesiveness accounts for the success of relatively traumatic surgical procedures such as iridectomy and iridocyclectomy, which are used mainly for tumors composed of highly cohesive spindle cells.

Although few ophthalmic surgeons have considered the possibility that enucleation may have detrimental rather than beneficial effects, general surgeons and experimental oncologists are concerned that various manipulations of the primary tumor (preoperative as well as intraoperative) might shower the venous or

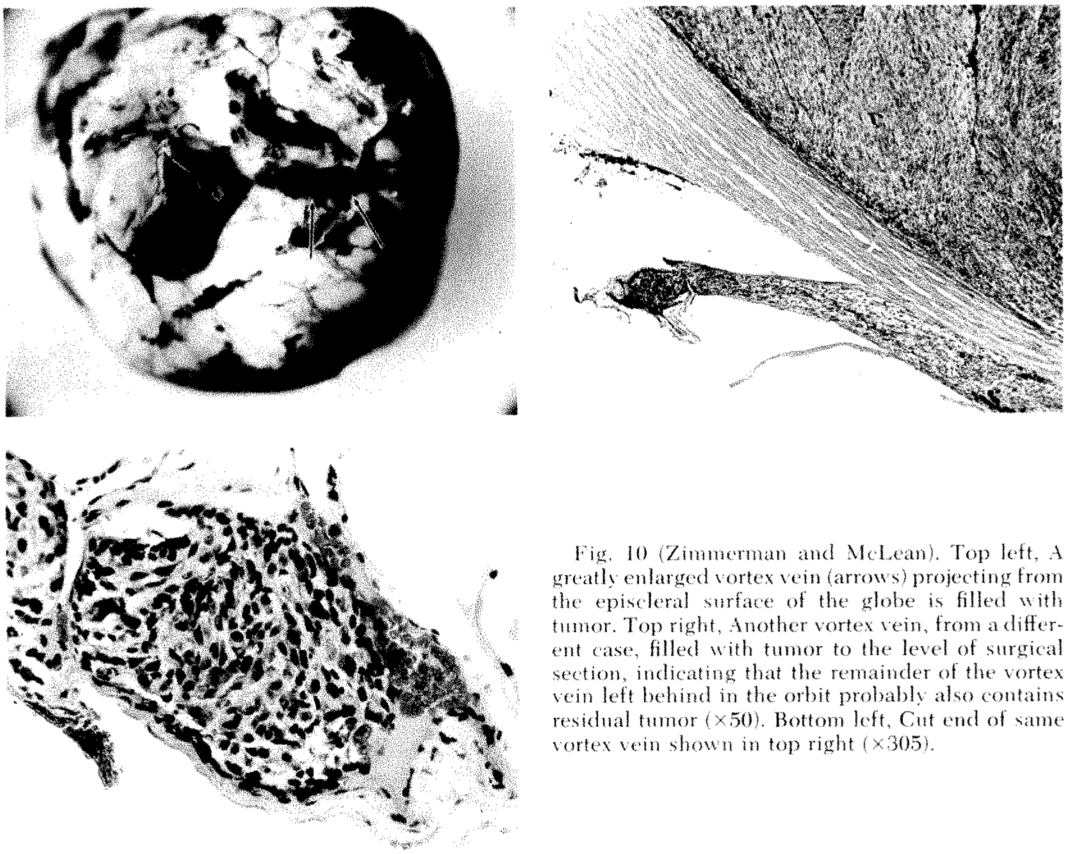


Fig. 10 (Zimmerman and McLean). Top left, A greatly enlarged vortex vein (arrows) projecting from the episcleral surface of the globe is filled with tumor. Top right, Another vortex vein, from a different case, filled with tumor to the level of surgical section, indicating that the remainder of the vortex vein left behind in the orbit probably also contains residual tumor (×50). Bottom left, Cut end of same vortex vein shown in top right (×305).

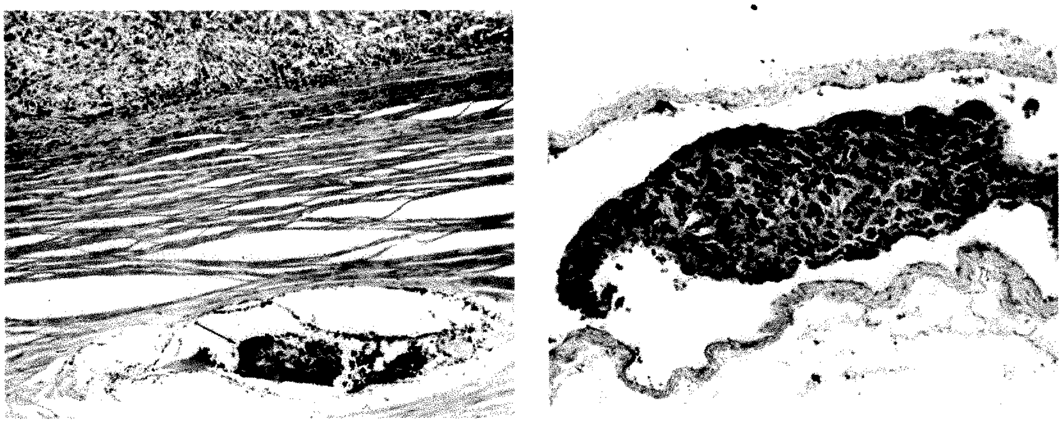


Fig. 11 (Zimmerman and McLean). In this case large tumor emboli were observed within vortex veins, both within a scleral canal (left), and in separate sections of a vortex vein attached to the globe (right). Left, Arrow indicates tumor embolus within an intrascleral vortex vein. Right, Exceptionally large tumor embolus in the separately sectioned extraocular segment of a vortex vein.

lymphatic circulations with tumor cells, thus overcoming the patient's natural threshold for resisting a "tumor-take" when neoplastic cells enter the circulation.[64,65,75-81] The term "iatrogenic tumor dissemination" is used to convey this possibility.[78] In general surgery the tendency for dissemination increases with increasing tumor size, approximately half of all clinical cancers having metastasized by the time of diagnosis. This high rate of metastatic disease before surgical intervention makes it much more difficult for general surgeons to know how much metastatic disease is iatrogenic. Nevertheless as Sugarbaker and Ketcham[78] stated:

> Iatrogenic tumor dissemination should be minimized by the judicious choice of biopsy, early venous and lymphatic ligation when feasible, en bloc resection technique, and gentleness in tumor manipulations.

The method of treatment of a tumor may affect the patient's immunologic defense mechanisms against it. Federman and co-workers[62,63] reported a rise in antibody levels following photocoagulation treatments. Even after apparent destruction of the tumor additional photocoagulation increases the antibody titer. The greatest rise in tumor-associated antibody levels followed cobalt plaque therapy. Enucleation, on the other hand, was associated with a fall in antibody levels. Federman and co-workers believe those methods of treatment that destroy rather than remove the tumor may autoimmunize by leaving necrotic tumor tissue in situ.

The experiments of Wexler and co-workers[64] support the thesis that blocking antibodies, which impair the host's ability to remove circulating cells and to prevent the development of metastatic lesions, are preferentially absorbed by the primary neoplasm. After the neoplasm is surgically resected, an excess of blocking antibodies develops, which by protecting tumor cells in the circulation, in the liver, or elsewhere accelerates the development of metastatic disease. Anesthetic agents and the surgical procedure itself may also play a significant immunosuppressive role that leads to an increased incidence of metastatic disease.[65] According to Lundy and co-workers,[65] in the face of an impaired tumor-specific immune response, a milieu for the establishment of distant metastasis is potentially present. In addition to the potential danger presented by tumor cells released into the circulation at the time of surgery, minute foci of tumor cells already implanted preoperatively may be given an increased chance to escape immune destruction and proliferate.

Griffiths and co-workers[81] hypothesized that fibrinolysis induced by the extensive operative procedures involved in the resection of colonic cancers may protect against the implantation of neoplastic cells disseminated during the operation. Perhaps much less significant fibrinolysis is induced during enucleation and there is, therefore, less protection against the development of metastasis.

Ophthalmologists will probably continue to use traditional methods until reliable data are available on which to base therapeutic recommendations. Institution of collaborative studies, national or international in scope, to test and compare various therapeutic regimens would promote a more rapid acquisition of basic information. Some studies already underway by individual investigators are concerned mainly with the treatment of small asymptomatic tumors, which are being observed without treatment by some investigators,[11,17,26,82] or by enucleation, radiation, photocoagulation, or excision by others.[15,21,27-29,45,82-88] Comparatively little has been done to improve the results that have been obtained by enucleation in the management of larger tumors. Vannas,[89] Sobánski and co-workers,[32-34] and Lommatzsch and Dietrich[90-91] irradiated the orbit after enucleation and claim significantly improved survival as compared with enucleation alone. Sobánski and co-workers [32-34] suggest that postoperative radiation damages or destroys tumor cells scattered into the surrounding tissues during enucleation. An argument against this hypothesis is that the frequency of postoperative metastatic disease greatly exceeds local recurrence in the orbit. An alternate hypothesis that we can advance from our histopathologic studies is that tumor cells dislodged into the venous drainage may get trapped temporarily in episcleral or intraorbital veins or both (Fig. 12). If enucleation is followed by either postoperative radiation therapy or by early exenteration, as recommended by Shammas and Blodi[92] for those cases in which extraocular extension is known to have occurred, localized tumor emboli and intravascular extensions would be eliminated.

Radiation before enucleation[93-95] seems even more logical. In their review of preoperative irradiation in head and neck cancers, Perez, Marks, and Powers[95] gave as one of their three indications for such use of radiation ". . . patients with relatively localized lesions in whom extensive tumor may contribute to dissemination of viable tumor cells at the time of surgery." According to these authors preoperative irradiation has been shown to have a sound biologic basis, and in most animal tumor systems, marked improvement in tumor control and survival has been demonstrated with a combination of radiation and surgical resection. In clinical experience with carcinomas of the paranasal sinuses, base of the tongue, tonsillar fossa, hypopharynx, and larynx, a preoperative dose of 3,000 to 5,000 rads has been associated with improved results but the absence of randomized clinical trials makes it less certain that the value of preoperative irradiation has been proven.[95] In 1943 Burch and Camp[96] reported on their attempt to improve prognosis by preoperative radiation. Although they had used only "a total dosage of 1,500 roentgens," they recorded "five-year cures" in two thirds of 21 treated patients, as compared with cures in only a little over one half of the nonirradiated patients. The authors, however, were not encouraged by their results, and A. B. Reese, in a discussion of their paper stated ". . . that irradiation of such tumors is

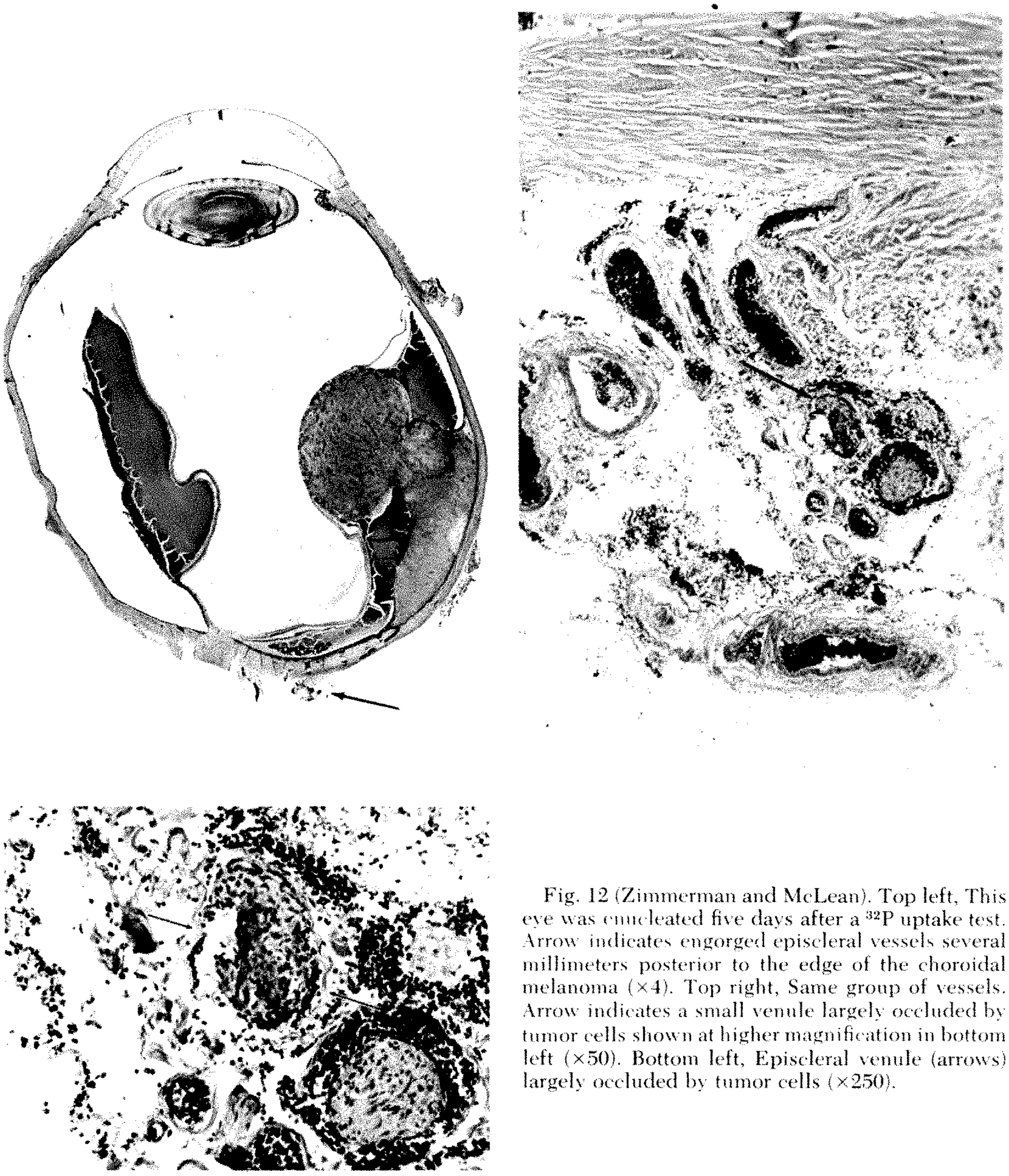


Fig. 12 (Zimmerman and McLean). Top left, This eye was enucleated five days after a ^{32}P uptake test. Arrow indicates engorged episcleral vessels several millimeters posterior to the edge of the choroidal melanoma (×4). Top right, Same group of vessels. Arrow indicates a small venule largely occluded by tumor cells shown at higher magnification in bottom left (×50). Bottom left, Episcleral venule (arrows) largely occluded by tumor cells (×250).

without benefit in regard to arresting the growth, preventing metastasis, or prolonging life."

To prevent dissemination of tumor cells, Fraunfelder and co-workers[58,59] and Wilson and Fraunfelder[60] freeze the entire tumor before the eye is enucleated, but they have not yet presented results for evaluation. These investigators report that careful handling of the ocular tissues and lifting maneuvers rather than pressure on the globe, prevent increases of intraocular pressure above 50 mm Hg before the tumor is completely frozen.

Frank LaPiana and Kenneth Kramer (personal communication, July 1978) used the same technique on several patients, and they have confirmed the observations of Fraunfelder and associates. They have refined the surgical procedure by using a pressure-stabilizing device that greatly reduces the magnitude of increases in intraocular pressure.[97,98] In this regard, it would be interesting to know more about the surgical technique used to achieve the outstanding results (ten-year mortality of 27%) reported by Benjamin and associates.[16]

As Lundy and co-workers[65] have suggested, it seems advisable to look prospectively at human protocols, starting immunotherapy before surgery in high-risk patients, and to avoid use of anesthetic agents or combinations that might be deleterious to the cancer patients. The adjunctive use of nonspecific immunepotentiators should be considered.

DISCUSSION

We agree with the following fundamental concepts: (1) a malignant tumor that has the potential to produce metastatic disease should be treated, and (2) surgical removal of the tumor-containing organ is one logical method of attempting to prevent death from metastasis, but we should document the validity of these concepts for each situation in which they seem applicable. Uveal melanomas require scientific evaluation in at least three different therapeutic situations. First, there is the question of how best to manage the comparatively small tumor discovered in an eye that is either asymptomatic or at least retains useful vision. Then, there is the most common situation, management of medium- to large-sized tumors associated with considerable loss of vision. Today, many ophthalmologists believe these cases cause no therapeutic dilemma because enucleation is indicated. Finally, there are the very large or diffuse tumors and those with extraocular extension that have the worst prognosis.

From this review of medical writings, unpublished observations made by us and by our colleagues in clinical ophthalmology and pathology, and our analysis of actuarial data derived from cases treated by enucleation, we cannot make logical recommendations concerning management of uveal melanomas because of our incomplete information as to the natural behavior of untreated tumors. A National or International Registry of Untreated Uveal Melanomas* should be established in an effort to ascertain: (1) the frequency and rapidity with which small, flat melanomas in asymptomatic eyes grow and produce secondary changes such as retinal detachment, uveitis, intraocular hemorrhage, secondary glaucoma, and the like; (2) the frequency and rapidity with which larger symptomatic tumors produce extraocular extension; and (3) the frequency and rapidity with which tumors of all sizes produce metastatic disease before any therapeutic interference with their natural course.

We agree with Char's[99] statement:

> A number of issues remain unresolved, and only by doing prospective studies, in a randomized manner when ethically feasible, can we better understand the natural history of choroidal melanomas and the effect of different therapeutic modalities on incidence of metastasis.

Long-term collaborative studies using randomized clinical trials should also be devised to evaluate and compare the relative efficacy of various adjunctive methods of treatment or modifications of routine enucleation, or both, such as: (1)

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*We do not recommend a moratorium on enucleation so as to be able to ascertain the natural history of untreated melanomas. Many other circumstances account for a small percentage of uveal melanomas remaining untreated. These cases should be systematically collected, documented, and followed up in a central Registry.

application of the "no-touch" concept before and during enucleation; (2) freezing of the tumors before and during enucleation; (3) use of pressure-regulating systems during enucleation; (4) administration of anticoagulants or drugs that inhibit platelet aggregation preoperatively and postoperatively; (5) preoperative or postoperative radiation therapy, or both; (6) preoperative or postoperative chemotherapy, or both; (7) preoperative or postoperative immunotherapy, or both; and (8) various combinations of the above.

SUMMARY

Uveal melanomas are slow growing tumors that may infiltrate out of the eye, mainly following ciliary vessels or nerves, or invade blood vessels, but only rarely do they produce metastatic disease before discovery and treatment. There are, however, large gaps in our knowledge of the frequency and rate of growth of small tumors to those of moderate or large size. We have estimated the mortality from untreated uveal melanomas to be 1% per year or less. Almost all information concerning prognostic factors has been gained from studies based on cases treated by enucleation. The same information is not necessarily applicable to untreated tumors. A study of deaths from metastatic uveal melanomas reveals a striking relationship to enucleation, regardless of whether one deals with cases in which the eye had been symptomatic for a long or short time. Following a low preoperative mortality of 1% or less, the annual mortality rises to about 4% during the first year and then peaks at 8 to 12% during the second year, tapering off during the next three to five years to return to a remarkably constant level of 1 to 2% thereafter.

We postulated that this relationship of peak mortality to enucleation is attributable mainly to an often overwhelming dissemination of tumor cells taking place during surgery or to a lowering of the host's immunologic defense mechanisms as a consequence of the operation, or both.

We urge the pooling of all available information gained from clinical studies made on untreated tumors and recommend prospective studies of a variety of other methods of treating uveal melanomas in addition to the standard enucleation.

ACKNOWLEDGMENTS

D. M. Albert, M.D., F. C. Blodi, M.D., P. Chretien, M.D., R. Foos, M.D., W. R. Green, M.D., F. Jakobiec, M.D., O. A. Jensen, M.D., G. Meyer-Schwickerath, M.D., G. Naumann, M.D., J. Shields, M.D., M. Smith, M.D., M. Vogel, M.D., and M. vonFricken, M.D., reviewed or translated some of the older published reports, or provided unpublished data.

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SURVIVAL RATES AFTER ENUCLEATION OF EYES WITH MALIGNANT MELANOMA

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Zimmerman, McLean, and Foster[1] recently questioned whether enucleation of eyes with malignant melanoma acclerated the dissemination of tumor cells, thereby increasing the risk of death. The evidence used in favor of such a hypothesis came mainly from mortality data published in 1962 by Paul, Parnell, and Fraker.[2] Reanalyzing these data, Zimmerman, McLean, and Foster[1] were particularly impressed with

> the rapid rise in mortality that follows enucleation, reaching a peak during the second post-enucleation year. Then during the ensuing several years the tumor death rate falls, reaching a low apparently constant level about six to seven years after enucleation.

Additionally they argued that few patients with primary melanoma of the uvea have metastases before surgery and that patients whose eyes are not enucleated often have a higher survival rate.

To evaluate whether the postsurgical trends in mortality described by Zimmerman, McLean, and Foster[1] were unique, we reanalyzed the data published by Paul, Parnell, and Fraker[2] and data gathered from other sources concerning uveal melanoma and other cancers.

RESULTS

Paul, Parnell, and Fraker[2] analyzed the mortality from all causes in patients registered with the Armed Forces Institute of Pathology (AFIP) who had an eye enucleated for malignant melanoma before January 1956. Of 3,385 available cases, "adequate follow-up data were procured in only 2,652." Their report provided a survivor curve after surgery for these 2,652 cases. From their survivor curve, we computed the risk of death in each year after surgery (Table 1).

These rates of death differed from those of Zimmerman, McLean, and Foster[1] because the denominator we used was the number alive at the beginning of the pertinent year after diagnosis, whereas they calculated the proportion dying each year, always using the total number having surgery as the denominator. Their computation showed the diminution of

TABLE 1
RISK OF DEATH IN EACH YEAR AFTER SURGERY AMONG THOSE STILL ALIVE AT BEGINNING OF YEAR.

Year After Surgery	Death Rates (deaths/100 alive at start of year)	Death Rates From Zimmerman, McLean, and Foster[1]
1	4.1	4.1
2	8.1	7.8
3	7.9	7.0
4	6.7	5.4
5	6.2	4.7
6	3.5	2.5
7	2.6	1.8
8	3.3	2.2
9	3.1	2.0
10	4.3	2.7
11	1.8	1.1
12	2.4	1.4
13	2.0	1.1
14	1.4	.8

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the original group of surgery patients by death each year, which does not measure the year-by-year risk of mortality.

For the first follow-up year both methods gave the same results. With increasing follow-up time, their proportion approach yielded lower values than we found. The difference between these methods becomes apparent when applied to the analysis of mortality in an elderly population. Whereas the risk of death was much higher for persons at age 100 years than at age 60 years, the proportion calculation would give the opposite impression. Even with our computation, however, a higher death rate was seen in years two to five than in years one or six to ten. After year ten, a further decrease was seen to levels of about 2%.

Comparison of different periods after surgery was difficult because the Paul, Parnell, and Fraker[2] data did not permit sampling errors to be computed for each year, although it was likely that long-term values had large sampling errors. Additionally, because of the large number of patients lost to follow-up there was reason to remain skeptical about the rates in later years. Finally, interpretation of the trend in mortality was complicated by the likelihood that the later rates were provided by patients whose ages were different than at time of surgery, both because of aging and varying survival rates in different age groups.

The observation that mortality tends to be higher in the early years after diagnosis of cancer is common. Ederer and Moss[3] contrasted the observed numbers of deaths to the expected numbers of deaths for men of the same age who had cancer of the tongue (Table 2). The ratios were markedly higher in years one and two after diagnosis than in years three through six. This was true moreover both for surgically treated cases as well as those treated by radiation, and was independent of whether metastasis had already taken place.

TABLE 2

MORTALITY OF MEN DIAGNOSED WITH LOCALIZED AND REGIONAL CANCER OF THE TONGUE TREATED BY SURGERY AND RADIATION, 1950-1957 (RATIO OF OBSERVED TO EXPECTED NUMBER OF DEATHS)

Treatment Follow-up Years	Mortality Ratio* Localized	Regional
	Surgery	
1,2	3.6	10.0
3-6	2.4	2.1
	Radiation	
1,2	5.8	12.2
3-6	2.6	3.8

*From Ederer and Moss.[3] Mortality ratios computed by dividing number of observed deaths by number of deaths expected for men of similar age composition in general population.

We investigated death rates for several tumor types, including malignant melanoma of the eye, for selected stages of disease and treatment modes (Table 3). The data came from four tumor registries whose cases were pooled and analyzed by the National Cancer Institute.[4] (These were the Connecticut Tumor Registry, the University of Iowa Hospitals, Charity Hospital of New Orleans, and the California Tumor Registry.) The pattern of peak mortality in the early years after diagnosis and lower rates five to ten years thereafter was common. For three of the columns in Table 3, the maximum rate of mortality was noted in the first year after diagnosis; in all others the maximum rates tended to occur one or two years later. When mortality for these three sites was examined by three month intervals in the first two years (Table 4), the pattern of peak mortality immediately after diagnosis and surgery was retained only in surgically treated stomach cancer patients. Complications from surgery may be the principal factor in this pattern.

The Cancer Institute rates for melanoma of the eye were similar in the first five

TABLE 3

DEATH RATES PER 100 PERSONS ALIVE AT START OF YEAR BY YEARS AFTER DIAGNOSIS FOR SELECTED SITES, STAGE OF DISEASE, AND MODE OF TREATMENT IN CASES DIAGNOSED FROM 1940 TO 1973

	Site/Stage/Treatment Modality									
Years After Diagnosis	Melanoma of the Eye/ Local/ Surgery (any)*	Melanoma of the Skin/ Local/ Surgery (any)*	Larynx/ Local/ Radiation Only	Larynx/ Local/ Surgery Only	Stomach/ Local/ Surgery (any)*	Cervix/ Local/ Radiation Only	Corpus/ Local/ Surgery (any)*	Corpus/ Regional/ Surgery (any)*	Breast/ Local/ Surgery (any)*	Breast/ Regional/ Surgery (any)*
1	4.30	4.54	14.61	7.73	25.30	9.34	4.58	28.59	3.82	12.56
2	8.08	8.49	13.83	8.49	18.56	12.77	5.41	19.57	5.58	17.56
3	6.28	8.52	11.90	7.88	17.10	8.25	4.54	12.55	6.24	15.77
4	9.01	7.16	7.85	7.26	12.46	6.67	3.67	9.38	6.32	14.66
5	6.40	6.90	7.47	5.83	9.52	4.81	3.18	5.99	6.22	12.46
Mean 6-10	6.12	4.15	8.29	6.58	7.43	3.60	3.09	5.49	5.49	9.63
Number of patients treated by indicated therapy	983	4,343	2,168	2,435	3,248	8,947	11,918	2,664	29,090	31,119
Percent at this site/stage treated by indicated therapy	92.74	95.91	38.60	43.32	66.80	65.60	80.03	49.06	95.24	80.56
Percent lost to follow-up over the ten-year period†	9.46	13.19	7.20	7.39	5.00	7.71	12.17	5.44	9.39	5.28

*Treatment modality surgery (any) includes surgery only, surgery + radiation, surgery + chemotherapy/hormones, and surgery + radiation + chemotherapy/hormones.

†Cases reported alive with date of follow-up before December 1973. National Cancer Institute, special tabulations of data from four tumor registries.[4]

TABLE 4

DEATH RATES PER 100 PERSONS BY QUARTER-YEARS (3-MONTH INTERVALS) AFTER DIAGNOSIS FOR THREE SELECTED PATIENT GROUPS IN CASES DIAGNOSED FROM 1940 TO 1973*

Months After Diagnosis	Site/Stage/Treatment Modality		
	Stomach/ Local/ Surgery (any)	Larynx/ Local/ Radiation Only	Corpus/ Regional/ Surgery (any)
3	11.93	2.23	6.03
6	4.29	3.83	9.73
9	4.73	4.37	9.40
12	6.45	4.87	7.54
15	6.07	4.43	6.22
18	5.04	3.57	5.11
21	4.99	2.96	5.44
24	3.96	2.54	4.48

*National Cancer Institute, special tabulations of data from four tumor registries.[4]

years to those of Paul, Parnell, and Fraker[2] but were higher in years six to ten. The increased rate in the fourth year was probably the result of the relatively small number of cases available for analysis. However the National Cancer Institute series did not lose as many to follow-up as the Armed Forces Institute of Pathology series and this may explain the decreasing rates of mortality in later years.

DISCUSSION

Zimmerman, McLean, and Foster[1] offer a number of arguments that lead them to speculate whether enucleation causes an increase in mortality in the early years after surgery. The major points made by them are: (1) few patients with primary melanoma of the uvea have metastases before surgery but metastasis is common after surgery; (2) mortality rates before surgery are low, increase sharply after surgery, and then decline; and (3) patients whose eyes are not enucleated often have higher survival rates as compared with those who do have surgery.

Alternative explanations, other than surgical effect, for each of these respective points are as follows:

1. Ocular tumors are often small at the time they become symptomatic or diagnosed. Metastases from such tumors to other areas of the body are likely to be even smaller and consequently may be difficult to diagnose. Additionally, uveal melanoma cases with distant metastasis before enucleation, although infrequent, are not nearly as rare in the National Cancer Institute series (2.5%) as they are in the Armed Forces Institute of Pathology files (0.4%).

2. With regard to the markedly increased risk of death in the Armed Forces Institute of Pathology cases after surgery, there are several reasons why one would not infer a causal relationship with enucleation. First, the data set cited[2] have a considerable proportion of patients lost to follow up. Second, the gradient in mortality is not as striking as in the report by Zimmerman, McLean and Foster,[1] because their method of computation of death rates makes rates in later years artifactually lower.

Even if an excess in mortality occurs in the early years after surgery, this may be the result of enucleation being done only after evidence of tumor growth. Thus enucleation is often coincident with an active phase of malignancy during which the rate of mortality is increasing. This pattern of increased mortality shortly after diagnosis is observed for cancers of many other sites. The pattern can be shown for nonsurgically treated diseases, and for those in which metastasis has already occurred. If surgical intervention causes this pattern for uveal melanoma, then there is cause for concern about this treatment for cancer generally. It seems to us more reasonable to infer that an active phase of progression causes the surgery, than that the surgery causes the disease to become more active.

3. Additionally, growth in the tumor either produces symptoms or indicates that the tumor is malignant. Tumors that don't grow and are not symptomatic are much less likely to be surgically removed. Thus, to conclude that untreated tumors have a favorable prognosis confuses cause and effect. In those patients who refuse surgery, it is important to consider that they are qualitatively different from those who accept surgery, because the physician may have equivocated in providing advice to the patient, and because the tumor was small or was not increasing in size. Additionally, asymptomatic patients are more likely to refuse surgery.

This analysis of survival rates in patients with uveal melanoma and other cancers produces no evidence to alter the existing pattern of treatment for uveal melanoma. Nonetheless current treatment should not go unchallenged. Thus the studies of Zimmerman, McLean, and Foster[1] stimulated interest in the study of alternative treatment strategies. It is difficult, however, to evaluate a therapy for which there is little variation in clinical practice. To make progress, treatment alternatives must be evaluated with well-designed clinical trials or we run the risk of continuing to support inferences with inadequate studies and clinical impressions. Until such clinical trials are conducted, treatment will be primarily motivated by the theory that it is in the patient's best interest to remove neoplastic tissue in the eye, as we do in other sites.

SUMMARY

We investigated the rates of mortality for several types of malignant melanomas for evidence that surgery accelerates metastasis. Additionally, we reanalyzed uveal melanoma survival rates from the Armed Forces Institute of Pathology. Our computations showed higher death rates in years two to five after diagnosis than in years one or six to ten. The same pattern of a peak mortality in the early years after diagnosis and lower rates six to ten years thereafter was seen in all tumor types studied. Our analysis of survival rates produced no evidence to alter the existing pattern of treatment for malignant melanoma of the uvea.

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RECURRENT ORBITAL AND METASTATIC MELANOMA IN A PATIENT UNDERGOING PREVIOUS GLAUCOMA SURGERY

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Although orbital extension of choroidal and ciliary body melanomas has been documented,[1,2] orbital recurrence after enucleation without histopathologic evidence of extrascleral extension is rare (0.4 to 0.7%).[1,2] Zimmerman, McLean, and Foster[3] speculate that enucleation may have an adverse rather than a beneficial effect with respect to the development of metastatic disease.

We describe herein a patient who underwent glaucoma filtering surgery shortly before enucleation for a large choroidal and ciliary body melanoma. The patient subsequently developed both local orbital recurrence and fatal metastatic disease.

CASE REPORT

A 61-year-old diabetic woman had a one-week history of sudden loss of vision in her right eye. Examination revealed an intraocular pressure (IOP) of 54 mm Hg in the right eye, a cloudy cornea, and a shallow anterior chamber. An inferior retrolental mass was interpreted as an organized hemorrhage. The left eye was normal. Attempts at controlling the right eye IOP medically were unsuccessful, and a Scheie filtering procedure with sector iridectomy was performed on Feb. 26, 1976. Postoperatively the anterior chamber remained flat and the IOP was high. The next week she was transferred here for further examination.

On examination, visual acuity was R.E.: no light perception and L.E.: 6/9 (20/30). The right eye was injected and the filtering surgery site was evident. The anterior chamber was shallow with a pigmented mass on the iris and in the angle inferonasally. A large nontransilluminating retrolental mass in the same quadrant was evident (Fig. 1). Intraocular pressure with the patient taking acetazolamide was R.E.: 41 mm Hg and L.E.: 11 mm Hg. We noted that no view was possible of the right posterior pole. The left eye was normal. There was a deep anterior chamber with an open iridocorneal angle of approximately 30 degrees by gonioscopy.

Results of diagnostic studies including chest x-ray, mammograms, cervical cytology (Pap smear), hemogram, liver enzymes, sedimentation rate, and liver-spleen scan were normal. Computed axial tomography with and without contrast revealed a large ocular tumor with no evidence of orbital mass or abnormality of the optic nerve or rectus muscles. Ultrasound examination confirmed the above findings.

The right eye was enucleated March 9, 1976. The conjunctiva was incised around the area of the filtering surgery to leave the bleb site attached to the globe. Histopathologic sections showed a complete retinal detachment and a large pigmented tumor originating from the ciliary body and choroid (Fig. 2). The melanoma was composed of both spindle and epithelioid cells. Tumor cells were seen around a nerve in the sclera, but no extrascleral extension of tumor was found. Multiple sections through the glaucoma filtering area revealed clumps

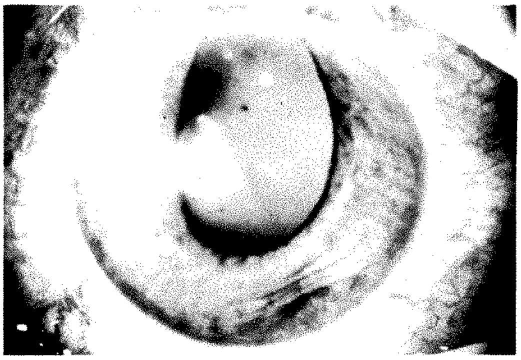

Fig. 1 (Singer and associates). The right eye after glaucoma filtering surgery. The anterior chamber is shallow and a pigmented mass is present on the iris and in the angle inferonasally.

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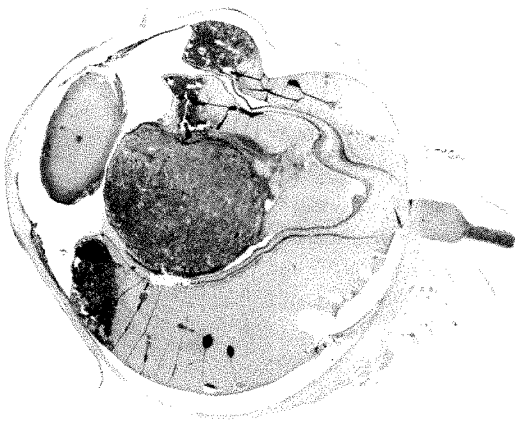

Fig. 2 (Singer and associates). The malignant melanoma originating from the ciliary body and choroid is composed of both spindle and epithelioid cells.

of pigmented debris in both the anterior chamber and in the filtration site, but no definite tumor cells could be identified. The postoperative course was uneventful and the final diagnosis was malignant melanoma of the mixed cell type of the choroid and ciliary body, with intrascleral extension.

In September 1978, the patient was again referred to us with a one-month history of difficulty with her prosthesis. Examination showed a small, firm black mass in the central portion of the right socket. Visual acuity in her left eye was correctable to 6/6 (20/20) and results of the examination of the left eye were normal. Biopsy of the pigmented right orbital mass showed malignant melanoma. Results of diagnostic studies including hemogram and chest x-ray were normal. Bone scan was equivocal and liver-spleen scan showed hepatomegaly, borderline splenomegaly, and multiple focal hepatic defects. Results of liver enzyme tests were abnormal and included the following: alkaline phosphatase, 131 mU/ml (normal < 95); gamma glutamyl transpeptidase, 279 IU/ml (normal < 63); serum glutamic oxaloacetic transaminase, 60 mU/ml (normal < 40); lactic dehydrogenase, 577 mU/ml (normal < 250). Computed axial tomography showed no evidence of intracranial metastases but examination of the right orbit revealed two masses that enhanced with contrast, suggesting melanoma.

The patient was treated with dacarbazine (DTIC). One course of therapy was completed before the patient's discharge; she returned for two additional treatments as an outpatient. She died in October 1978. An autopsy was not performed.

DISCUSSION

A review of 1,872 cases of malignant melanoma of the choroid and ciliary body on file in the Registry of Ophthalmic Pathology showed histopathologic evidence of extrascleral extension in 13%.[2] Although orbital recurrence is frequent (18%) in those cases with extrascleral extension, it is extremely rare (0.7%) in those without extrascleral extension.[2] These findings are confirmed by the studies of Shammas and Blodi,[1] with histopathologic evidence of orbital extension occurring in 45 of 432 cases (10.4%) of choroidal and ciliary body melanoma. Local orbital recurrence is 23% in these 45 cases with evidence of extrascleral extension, but in only one of 263 cases (0.4%) with no orbital extension.

In patients with orbital recurrence without evidence of extrascleral extension, there has been no attempt to establish the existence of extension by making additional histopathologic sections.[1,2] In our case, numerous serial sections were made throughout the entire globe, which showed intrascleral extension only along a nerve. Therefore, it could be postulated that tumor cells spread through the glaucoma filtering site were responsible for the local orbital recurrence of melanoma.

A new philosophy regarding the management of an eye containing a melanoma has been proposed by Fraunfelder[4] and supported by Zimmerman, McLean, and Foster.[3] This philosophy evolved after the realization that most malignant tumors continuously shed cells into the blood stream and that showers of cells occur with manipulation of the tumor, for example, in surgery. Clumps of malignant cells have been found in the peripheral bloodstream during or immediately after enucleation in patients with choroidal malignant melanomas.[5] Experimental evidence supports the concept that the incidence of metastases was directly proportional to the number of malignant cells released into the circulation.[6] Zimmerman, McLean, and Foster[3] recently speculated that after enucleation for uveal melanoma, approximately two thirds of the fatalities could be attributed to dissemination of tumor emboli at the time of enucleation. A technique for removal of tumor bearing tissues referred to as "no touch" has been used clinically in cancer of the colon,[7] and experimentally for intraocular malignant melanoma.[4] Our patient did not have evidence of metastatic disease at the time of enucleation. The dissemination of tumor emboli could have occurred at many other times: before the acute glaucomatous attack, at the time of glaucoma filtering surgery, during the two-week interval after filtering surgery before enucleation, at the time of enucleation, or after enucleation secondary to local orbital recurrence.

The role of the glaucoma filtering surgery in the development of metastatic disease in our patient remains speculative. Filtering surgery results in more manipulation than routinely occurs before enucleation for a melanoma. Showers of melanoma cells into the aqueous humor leaving the eye via the filtering site is possible.

That the acute glaucomatous attack occurred in an eye containing a large melanoma was not known at the time of filtering surgery. However, the diagnosis of a secondary angle-closure glaucoma might have been suspected because the fellow eye had open angles on gonioscopy and deep anterior chamber. Although the glaucoma filtering surgery in this patient could have been the source of both the local orbital recurrence and fatal metastatic disease, this was not proven histopathologically.

SUMMARY

A 61-year-old woman had unilateral acute angle-closure glaucoma and subsequently underwent a Scheie filtering operation. After a two-week interval, a large choroidal and ciliary body melanoma in the glaucomatous eye was found. No evidence of metastatic disease was found and the eye was enucleated. Histopathologic sections did not show extrascleral extension. Two and one-half years later the patient developed local orbital recurrence of the melanoma and fatal metastatic disease. We hypothesized a possible causal relationship between the filtering surgery and the local and systemic recurrence of the tumor.

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COMPARISON OF RADIOACTIVE PHOSPHORUS (^{32}P) UPTAKE TEST IN COMPARABLE SIZED CHOROIDAL MELANOMAS AND HEMANGIOMAS

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Since 1970, the use of the radioactive phosphorus (^{32}P) uptake test in the differential diagnosis of intraocular tumors has gained wide acceptance. Early and valid criticisms of the procedure resulted in rigorous technique, particularly with regard to accurate localization of the lesion. Since the report of Hagler, Jarrett, and Humphrey[1] in 1970, several authors have stressed the accuracy of the procedure in differentiating benign from malignant lesions.[2-5] Although uncommon, choroidal hemangiomas occasionally are difficult to distinguish from choroidal melanomas, particularly amelanotic melanomas. From 5 to 10% of eyes enucleated with the mistaken diagnosis of choroidal malignant melanoma prove histologically to contain a hemangioma, rather than a melanoma.[6,7] Although the ^{32}P uptake test is usually negative in cases of choroidal hemangioma,[2,3,8,9] recent published reports have pointed out that false-positive results do occasionally occur.[5,10] A persistent question in distinguishing between choroidal hemangioma and melanoma is whether the negative ^{32}P results usually obtained with hemangiomas have been related to their small size; that is, hemangiomas give negative results because they are usually smaller in size than melanomas. We report herein the evaluation of the ^{32}P results in comparable sized hemangiomas and melanomas to determine whether there is a significant difference in ^{32}P uptake in these lesions that are size matched.

SUBJECTS AND METHODS

We reviewed the last 559 patients on whom the ^{32}P uptake test was performed here. The tests were performed between February 1971 and June 1977. Of these cases, there were 20 choroidal hemangiomas, with follow-up data ranging from one to six years. Accurate size measurements were available in 19 of these tumors. All of the tumors ranged in size from 3 × 3 × 2 mm to 8 × 8 × 4 mm. In one case the eye was enucleated and histologic sections of the tumor confirmed the diagnosis of hemangioma. Based on clinical findings, fluorescein angiography, ultrasonography, and careful follow-up examination, we believe that the remaining lesions are choroidal hemangiomas.

The ^{32}P series was then reviewed for those melanomas that were of a size comparable to the hemangiomas. In contrast to the choroidal hemangiomas, these lesions were histologically documented after enucleation. Like the choroidal hemangiomas, these 21 tumors all ranged from 3 × 3 × 1 mm to 8 × 8 × 4 mm.

A rough estimate of the size of these tumors was obtained by taking the product of their dimensions (height times base area). This is not the exact volume of the tumor, but merely a figure for comparative measurement. The average size measured in this manner for hemangiomas was 109 mm^3, whereas that of the malignant melanomas was 97 mm^3. Additionally, the actual average height of the choroidal hemangiomas was 2.5 mm whereas that of the malignant melanoma was 2.2 mm.

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In most instances, ultrasonography confirmed the clinical estimation of size.

Results were obtained by using both the detector ocular tumor unit (EON Corporation) and the solid state semi-conductor unit (Technical Associates).[11] Although the results obtained were similar with both instruments, we only report data from the EON unit.

Because all lesions were located posteriorly, it was necessary to perform a conjunctival incision for precise localization. Four counts were taken over the lesion and four control counts over the opposite quadrant of the same eye using a previously published technique.[11]

A positive ^{32}P uptake test was defined as an uptake of 50% or greater over the lesion as compared to a control area, for reasons listed in a previous publication.[3]

RESULTS

The results of the ^{32}P uptake test in choroidal hemangiomas and comparable sized choroidal melanomas are shown in Table 1. The average percent uptake over the lesion compared to the control area was 160% in choroidal melanomas (Table 2). Of the choroidal melanomas tested there were no false-negative results. The average percent uptake over the lesion as compared to the control area was 23% in choroidal hemangiomas (Table 2). There were two false-positive results.

DISCUSSION

The criticism that melanomas give positive results and hemangiomas give negative results occurred because the reported series[1,3,4] were comparing small hemangiomas to melanomas of all size groups. Our study clearly shows that the ^{32}P uptake results are significantly lower in choroidal hemangiomas than in comparable sized melanomas. However, cases have been described in which the ^{32}P uptake test will give false or misleading results. Cox[10] reported three cases in which false-positive results occurred with choroidal hemangiomas. However, clinicians have removed eyes because of the suspicious behavior of hemangiomas despite a negative ^{32}P uptake test that correctly indicated the benign nature of the lesion.[3,9] In our series, one eye was enucleated because progression of the associated retinal detachment aroused suspicion that the lesion might be a melanoma. The ^{32}P uptake in this case was negative and the histologic findings showed it to be a hemangioma.

Two choroidal hemangiomas gave false-positive results of 89 and 63%. Because of the clinical appearance, however, the lesions were observed and showed no clinical evidence of growth. No melanomas gave false-negative results. The overall results indicate a significant difference in the ^{32}P uptake with the two tumors and suggest that the test does play a helpful role in most cases.

Although the test appears to be reliable in differentiating small melanomas from choroidal hemangiomas, the procedure does have definite limitations in small tumors, especially in the differentiation of small choroidal melanomas from large choroidal nevi.[5] There have been a number of recent cases in which eyes were enucleated because of a positive ^{32}P uptake test and the lesion proved histologically to be a benign nevus.[12,13] In the differentiation of choroidal melanoma from hemangioma, however, the test is more reliable.

Not all patients with choroidal hemangiomas undergo a diagnostic ^{32}P uptake test. In most cases, a thorough clinical evaluation combined with ultrasonography will establish the correct diagnosis. Most of these cases were seen before our acquisition of the current A-scan units, which are highly accurate in differentiating choroidal hemangioma from melanoma.[14] Although we initially per-

TABLE 1

^{32}P UPTAKE RESULTS OF 40 CASES OF MELANOMAS AND HEMANGIOMAS IN INCREASING TUMOR SIZE*

Case No.	Tumor Size	Percent ^{32}P Uptake	Diagnosis
1	9	180	MM
2	18	12	CH
3	24	59	MM
4	27	10	CH
5	30	119	MM
6	32	18	CH
7	36	116	MM
8	38	0	CH
9	40	89	CH
10	50	48	CH
11	54	70	MM
12	72	24	CH
13	72	34	CH
14	72	−9	CH
15	72	53	MM
16	72	221	MM
17	72	90	MM
18	72	156	MM
19	72	170	MM
20	75	12	CH
21	84	90	MM
22	90	289	MM
23	90	355	MM
24	90	86	MM
25	108	39	CH
26	108	285	MM
27	112	120	MM
28	113	78	MM
29	135	33	CH
30	147	17	CH
31	147	11	CH
32	169	36	CH
33	172	0	CH
34	192	8	CH
35	192	109	MM
36	192	206	MM
37	196	83	MM
38	256	63	CH
39	256	0	CH
40	256	417	MM

*MM designates malignant melanoma, CH, choroidal hemangioma.

formed the ^{32}P uptake test on a number of choroidal hemangiomas, we now restrict its use to cases in which the clinical differentiation from choroidal melanoma is particularly difficult. Although the choroidal melanomas in this series were enucleated, our philosophy regarding management of such cases has changed. We do not enucleate most small melanomas but rather manage them by close obser-

TABLE 2

SUMMARY OF ^{32}P UPTAKE FINDINGS IN 19 CASES OF CHOROIDAL HEMANGIOMAS AND 21 CASES OF MALIGNANT MELANOMAS*

	Average Percent ^{32}P Uptake Results
Choroidal Hemangioma	23
Choroidal Melanoma	160

*$P<.002$.

vation. If they are highly suspicious, however, we manage them with photocoagulation, eyewall resection, or cobalt plaque.[15]

One case illustrates the diagnostic benefits of ^{32}P uptake test in differentiating choroidal hemangiomas and melanomas. A 26-year-old woman consulted her ophthalmologist because of decreasing vision. The diagnosis was made of a juxtapapillary choroiditis and she was treated with retrobulbar corticosteroid injections. She improved for a short time, but because of recurring symptoms sought help elsewhere. A subretinal neovascular membrane was subsequently diagnosed and the patient was treated with photocoagulation. On re-examination the diagnosis was changed to choroidal hemangioma and treatment with photocoagulation was continued. The symptoms persisted and she was referred to us, where the diagnosis was choroidal hemangioma. However, a ^{32}P uptake test was markedly positive, and enucleation confirmed the diagnosis of malignant melanoma.

SUMMARY

We compared 19 choroidal hemangiomas and 21 choroidal melanomas of comparable size as to the percent of radioactive phosphorus ^{32}P uptake. The results were significantly lower in choroidal hemangiomas than in comparable sized melanomas.

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THE ULTRASONOGRAPHIC AND RADIOLOGIC FEATURES OF A HISTOLOGICALLY PROVEN CASE OF ALVEOLAR SOFT-PART SARCOMA OF THE ORBIT

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Primary alveolar soft-part sarcoma of the orbit is extremely rare with only a few cases reported.[1,2] In a review of 53 patients with this tumor, Lieberman and associates[3] had no cases with orbital involvement. We report herein the ultrasonographic and radiologic features of a histologically proven case of alveolar soft-part sarcoma of the orbit.

CASE REPORT

An 18-year-old woman had a dilated epibulbar vessel on the temporal aspect of her right eye and an increase in prominence of the right eye, noted over a two-week period. A slight proptosis of the right eye was noted, 2 mm by the Hertel exophthalmometer, and thyroid function tests were done. The serum triidothyronine (T_3) was 42.3 ng/dl (normal 35 to 45 ng/dl). The serum thyroxine (T_4) measured 3.3 μg/dl (normal 4.6 to 11.6 μg/dl). The thyroid stimulating hormone was 3 μIU/dl (significant if over 10 μIU/dl). The results of these tests were within normal limits. The patient returned in two weeks complaining of sharp pain behind the right eye. A progression of the proptosis as well as an increase in the vascular congestion was noted and the patient was referred to us.

When seen by us, the patient was healthy and complained only of a lethargy of recent onset. Medical history was noncontributory. Family history revealed that three maternal uncles had died of various carcinomas.

Visual acuity was R. E.: 6/9 (20/30) and L. E.: 6/6 (20/20). External examination of the right eye revealed a proptosis of 3 mm as measured by a Hertel exophthalmometer (Fig. 1). The proptosis was nonpulsatile and nonreducible. Dilated episcleral vessels were noted temporally on the right eye (Fig. 2). The cranial nerves were intact. The fundi, examined by indirect ophthalmoscopy, were normal.

Water immersion ultrasonography indicated a 1 × 1 × 1.5-cm irregular orbital mass. The lesion extended along the lateral and superior wall of the

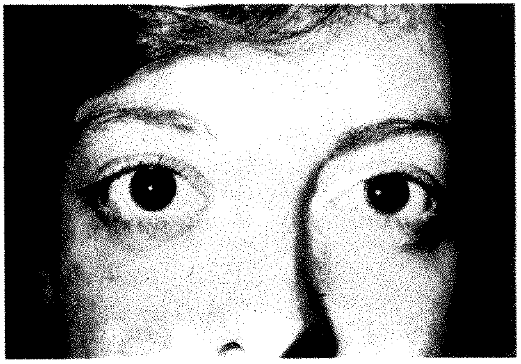

Fig. 1 (Grant and associates). Right eye, clinical appearance showing proptosis as indicated by the widening of the palpebral fissure.

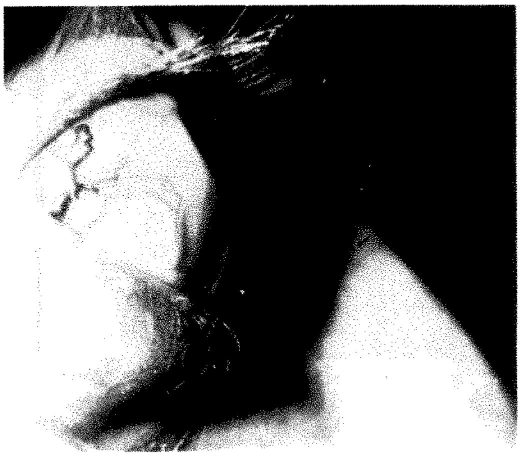

Fig. 2 (Grant and associates). Right eye, dilated episcleral vessels noted temporally.

From the Oncology Unit of the Retina Service (Drs. Shields, Grant, and Horowitz), and the Oculoplastic Service (Dr. Flanagan), Wills Eye Hospital, Philadelphia, Pennsylvania. This study was supported in part by the Retina Research and Development Foundation, Philadelphia, and the Pennsylvania Lions Sight Conservation and Eye Research Foundation, Inc. Dr. Grant is a Fellow of the E. A. Baker Foundation for the Prevention of Blindness.

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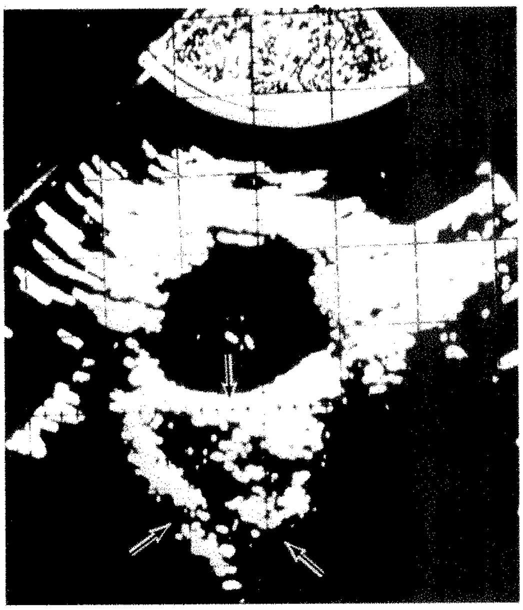

Fig. 3 (Grant and associates). Water immersion B-scan ultrasonogram shows a large retrobulbar mass lesion (arrows).

right orbit to the orbital apex. The mass exhibited moderate internal echoes and good sound transmission (Fig. 3). Contact B-scan ultrasonography with the Bronson-Turner unit confirmed the presence of a mass lesion that had few acoustic interfac-

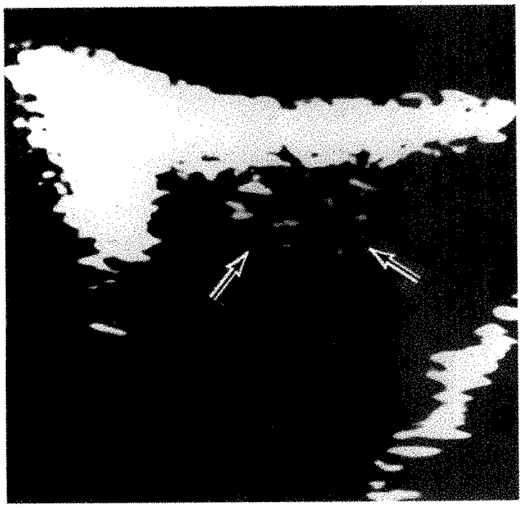

Fig. 4 (Grant and associates). Contact B-scan ultrasonogram shows the mass posterior to the scleral echo. A few internal reflections are noted (arrows).

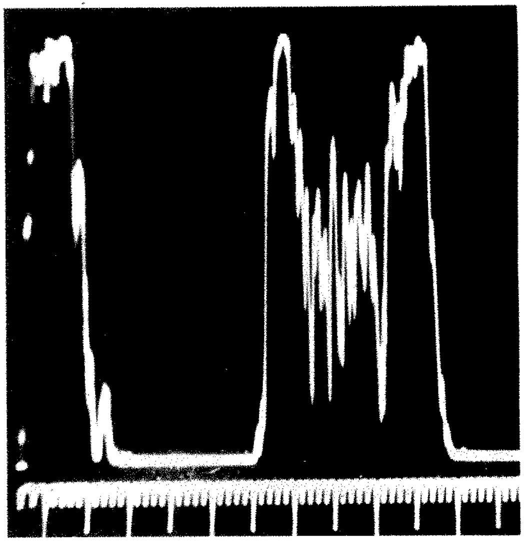

Fig. 5 (Grant and associates). Contact A-scan ultrasonogram shows a retrobulbar mass that exhibits moderate internal reflectivity, minimal attenuation and good transmission.

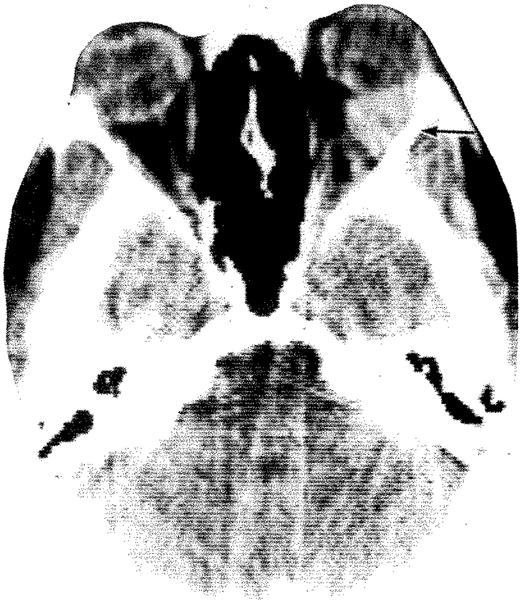

Fig. 6 (Grant and associates). Computed axial tomogram shows the presence of a mass lesion (arrow) filling the retrobulbar space and displacing the optic nerve medially.

es (Fig. 4). A-scan ultrasonography of the lesion showed minimal sound attenuation and moderate internal relfectivity (Fig. 5).

Skull series and tomograms were negative for bony erosion. Orbital venography showed a medial and downward displacement of the right superior ophthalmic vein. Computed axial tomography confirmed the presence of a mass located in the retrobulbar space on the right side displacing the optic nerve medially and producing a proptosis of the globe (Fig. 6).

A diagnosis of cavernous hemangioma of the orbit was considered and the patient was observed without treatment. Within two weeks, the patient returned complaining of increased pain. A further proptosis of the globe and increased prominence of the episcleral vessels were noted. Visual acuity was decreased to 6/21 (20/70) and could not be improved.

Carotid angiography was obtained before surgical exploration in order to identify possible large arterial feeder vessels that might indicate an arteriovenous malformation. This study showed a tumor blush but no major vessels (Fig. 7).

The right orbit was exposed by using the Kronlein approach with removal of the lateral orbital wall. The orbit was filled with a diffuse, noncohesive tumor with involvement of the orbital apex. The tumor was removed subtotally and submitted for histopathologic examination.

Low-power microscopy showed the lesion to be deeply basophilic and of uniform pattern throughout with an alveolar (organoid) arrangement of cells (Fig. 8). Under higher power, the lesion was noted to consist of large polygonal cells each with vesicular nucleus and prominent nucleolus (Fig. 9). This tumor also showed the presence of fine PAS-positive diastase resistant cytoplasmic granules. The histologic diagnosis was alveolar soft-part sarcoma.

A total of 6,000 rads in fractionated doses was given to the right orbit over a six-week period. Subsequent follow-up has shown a regression of the proptosis and alleviation of the patient's symptoms.

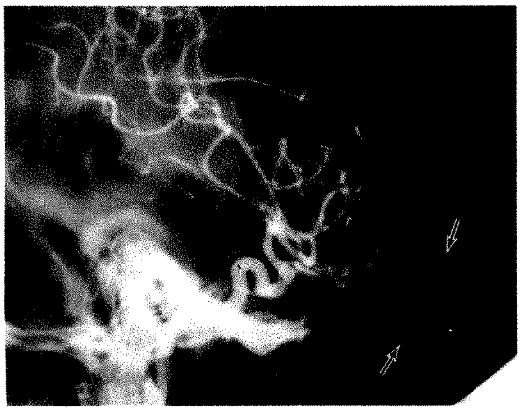

Fig. 7 (Grant and associates). Right internal carotid angiography shows the presence of an orbital tumor blush (arrows).

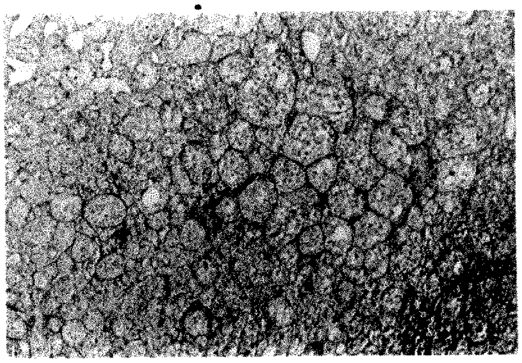

Fig. 8 (Grant and associates). Low-power photomicrograph showing the alveolar arrangement of cells (PAS, ×60).

DISCUSSION

Alveolar soft-part sarcoma is a tumor of unknown histogenesis that has been the subject of controversy for many years. Abrikossoff[4,5] described the features of a specific tumor having an organoid structure composed of cells with a granular cytoplasm. He named the tumor the granular cell myoblastoma and described both a benign and malignant form. Of the originally described cases, those malignant tumors subsequently recognized as rhabdomyosarcomas were deleted.[6] However, tumors that we now recognize as

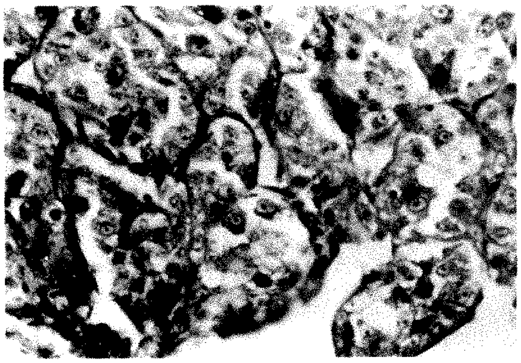

Fig. 9 (Grant and associates). High-power photomicrograph shows the vesicular nuclei and prominent nucleoli. (PAS, ×400).

alveolar soft-part sarcoma and malignant nonchromaffin paraganglioma continued to be unrecognized as specific entities and were included with granular cell myoblastoma.

It was not until 1952 that Christopherson, Foote, and Stewart[7] promoted the alveolar soft-part sarcoma as a distinct histopathologic entity. In 1963 Nirankai, Green, and Chaddah[8] described the nonchromaffin paraganglioma as a separate entity.

The recent studies of Fisher and Reidbord[9] and Shipkey and associates[10] have suggested differences in the granular ultrastructure of the alveolar soft-part sarcoma, malignant nonchromaffin paraganglioma, and the granular cell myoblastoma. Most pathologists now agree that these are different tumors; however, there is still no agreement as to the histogenesis.

Sobel, Marquet, and Schwarz[11] have provided evidence for a common neural precursor cell for both the granular cell myoblastoma and schwannoma. This is not the case for the alveolar soft-part sarcoma in which the precursor cell type is still controversial.

Fisher and Reidbord[9] believe that the alveolar soft-part sarcoma is a form of rhabdomyosarcoma. They believe that the granules represent Z-band material. This view is supported by the work of Marshall and Horn.[12]

Unni and Soule[13] stated that the crystalloids seen are membrane bound, and most likely represent secretory products, and are not myogenic in origin. They believe that the general structure of the alveolar soft-part sarcoma more closely resembles that of the nonchromaffin paraganglioma. This view is supported by Welsh and associates.[14]

Although the uncertainty as to the histogenesis continues, there is agreement that the prognosis for patient survival is poor. Altamirano-Dimas and Albores-Saavedra[1] reported two cases of alveolar soft-part sarcoma in which one patient died as a direct result of the disease. Lieberman and associates[3] reviewed 53 cases of alveolar soft-part sarcoma and noted an approximate 47% five-year survival rate. Two thirds of their cases occurred in females. A lateralization of tumor location was noted with 67% occurring to the right of midline. The average age of onset in females was 20 years. Although none of these cases occurred in the orbit, the statistics correlate well with our case.

SUMMARY

An 18-year-old woman had a rapidly progressive proptosis, vascular congestion, and orbital pain. Ultrasonographic and radiologic tests supported the diagnosis of a solid vascularized mass that proved histologically to be an alveolar soft-part sarcoma of the orbit. Uncertainty as to the histogenesis of alveolar soft-part sarcoma still exists, but prognosis for patient survival is poor.

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OPHTHALMIC MINIATURE

We were very sorry to part from him; indeed, Good was so moved that he gave him as a souvenir—what do you think?—an eye-glass. (Afterwards we discovered that it was a spare one.) Infadoos was delighted, foreseeing that the possession of such an article would enormously increase his prestige, and after several vain attempts actually succeeded in screwing it into his own eye. Anything more incongruous than the old warrior looked with an eye-glass I never saw. Eye-glasses don't go well with leopard-skin cloaks and black ostrich plumes.

H. Rider Haggard
King Solomon's Mines, 1885

ANTERIOR SEGMENT METASTASES FROM AN OVARIAN CHORIOCARCINOMA

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Most tumor metastases to the eye occur in the posterior uvea. Data accumulated from several series[1-3] indicate that only about 10% of ocular metastases occur in the anterior segment. The most common sites of the primary lesions are the breast and the lung.

Choriocarcinoma is a rare malignant trophoblastic tumor that originates in the female uterus and ovary and in the male testis. Rarely, extragenital primary choriocarcinomas have been described.[4,5] Gestational and nongestational types of choriocarcinoma are recognized, but gestational choriocarcinomas are associated with pregnancy, particularly pregnancy in which a hydatidiform mole occurs. Nongestational choriocarcinomas derive from germ cells of the testis or ovary.[6] Before the advent of chemotherapy choriocarcinoma was fatal in over 85% of cases. With the introduction in 1955 of methotrexate therapy for this tumor the outlook changed dramatically.[7] Currently, a number of therapeutic agents, used singly or, more frequently, in combination, have produced clinical cures in as many as 98% of female patients whose cases are diagnosed early and treated vigorously, even in the presence of metastases.[8]

Sixteen cases of choriocarcinoma metastatic to the eye have been reported.[5,9-23] We report herein an unusual case that documents metastatic choriocarcinoma solely to the anterior segment of the eye. We were able to make a tissue diagnosis of the ocular lesion early in its course, and were able to document the effect of therapy through clinical follow up and eventual histopathologic examination of the globe.

CASE REPORT

A 28-year-old woman (gravida 2, para 2) was admitted to the hospital for the first time in January 1975 at age 25 years with a pelvic mass and markedly increased human chorionic gonadotrophin titers of 5000 mIU/ml of serum determined by radioimmunoassay (normal by this method, <1 mIU/ml). Her last menstrual period had been in August 1974. Her previous pregnancies had been unremarkable and she had never had a spontaneous abortion. A laparotomy was performed for diagnosis and the ovarian mass was resected. The tissue diagnosis was choriocarcinoma. No evidence of ectopic pregnancy was found, and teratomatous elements were not present within the tumor. Chemotherapy with methotrexate and dactinomycin (Actinomycin D) was initiated and the human chorionic gonadotrophin levels decreased to <5 mIU/ml, but still were slightly higher than the normal range. After four months they precipitously rose again to 300 mIU/ml. A chest x-ray revealed a mass in the lower lobe of the right lung. Cyclophosphamide was added to the therapeutic regimen. The pulmonary mass decreased in size radiographically and the human chorionic gonadotrophin levels fell transiently to within the normal range, but then began to rise again. In November 1975, the patient underwent a right lower lobectomy. Histopathologic examination revealed necrotic tumor. Subsequently, she developed central nervous system symptoms. Diagnostic evaluation indicated a mass in the right frontal lobe. A craniotomy was performed and the tumor was resected. Over the next few months human chorionic gonadotrophin levels decreased to the normal range where they remained for nearly five months. In late 1976, the patient developed an anterior uveitis in the left eye that was initially treated with topical corticosteroids and atropine. Initially, no metastatic lesions were visible in the eye although it was suspected that these were responsible for the uveitis when no response to corticosteroids was observed and the human chorionic gonadotrophin levels rose to

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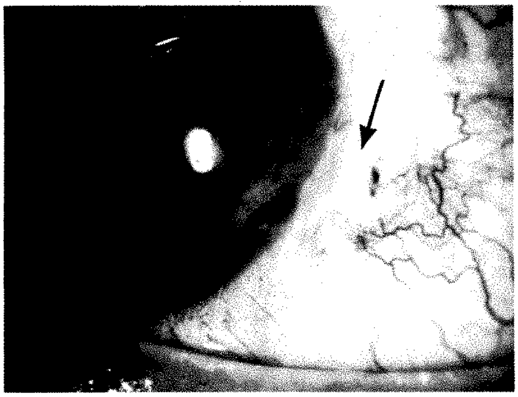

Fig. 1 (Frank and associates). Left eye two weeks before biopsy of conjunctival nodule (May 1977), showing a hemorrhagic mass in the anterior chamber and a subconjunctival nodule (arrow) apparently extending from it.

35 mIU/ml. By May 1977, a mass was visible in the inferonasal quadrant of the anterior chamber of the left eye and a second mass, which appeared to have extended from it, was visible subconjunctivally in the same quadrant (Fig. 1). Results of examination of the fundus, including indirect ophthalmoscopy with scleral depression were negative. Biopsy of the subconjunctival mass was done in June 1977 and histopathologic examination revealed a markedly vascular and hemorrhagic tumor (Fig. 2) composed of two cell types, consistent with the cytotrophoblastic and syncytiotrophoblastic elements of choriocarcinoma (Fig. 3). The cytotrophoblastic cells were large, polygonal cells, with pale-staining-to-clear cytoplasm, distinct cellular borders, and large, vesicular nuclei with prominent nucleoli (C, Fig. 3). The syncytiotrophoblastic cells had deeply staining eosinophilic cytoplasm and hyperchromatic nuclei. Their cell borders were indistinct and at times the cells had a tendency to form a multinucleated syncytium (S, Fig. 3).

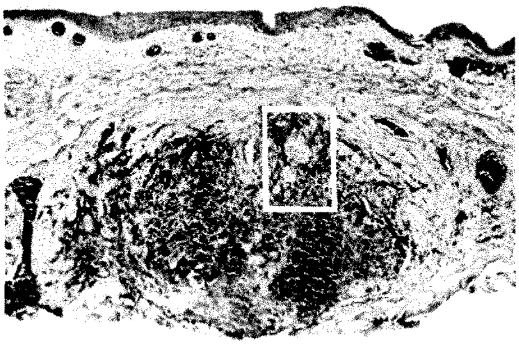

Fig. 2 (Frank and associates). Left eye, section of biopsy specimen showing a highly vascularized and hemorrhagic mass in the substantia propria of the bulbar conjunctiva. The area enclosed by the white rectangle is enlarged in Figure 3 (hematoxylin and eosin, ×95).

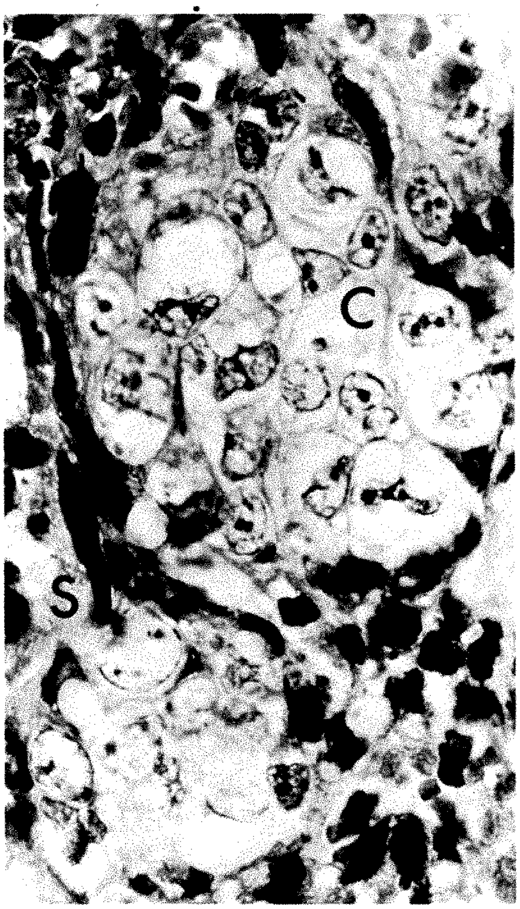

Fig. 3 (Frank and associates). Enlargement of the area within the rectangle in Figure 2 showing cytotrophoblastic cells (C), and syncytiotrophoblastic cells (S). (hematoxylin and eosin, ×630).

After surgery, visual acuity was 6/6 (20/20) in the right eye and 6/30 (20/100) in the left eye with moderate flare, cells, and posterior synechiae present. Gonioscopy revealed several apparent tumor nodules in the chamber angle, and ultrasonography showed a mass in the anterior segment but no abnormalities posteriorly. Ophthalmoscopic examination also continued to show no lesions in the posterior segment. Multiple photocoagulation treatments with the argon laser were given using the slit-lamp delivery system and the Goldmann gonioscopic lens to ablate tumor nodules in the chamber angle. In August 1977, radiation treatment to bulky portions of tumor located in the ciliary body was begun. The treatment consisted of 25 fractionated doses of radioactive isotope of cobalt (^{60}C) given over a period of 38 days; 200 rads per dose were given, bringing the total to 5000 rads. The patient continued to receive topical corticosteroids and atropine. There appeared to be some shrinkage of the tumor, and over the next three months, human chorionic gonadotrophin levels decreased to within the normal range. In October 1977, seizures developed and another cerebral metastasis was found. Systemic chemotherapy with methotrexate, dactinomycin, cyclophosphamide, and doxorubicin HCl (Adriamycin) was re-administered. Additionally, subconjunctival methotrexate was administered in November 1977 in an attempt to destroy residual ocular tumor. The uveitis persisted and the eye became painful and soft. Visual acuity decreased to 6/120 (20/400). Corneal epithelial defects and a vascular pannus were noted. Human chorionic gonadotrophin levels rose again to over 100 mIU/ml and the chemotherapeutic regimen was altered to include dactinomycin, vincristine, bleomycin sulfate, and cisplatinum.

By March 1978, vision had decreased to light perception and the eye remained soft and painful. Enucleation was performed on April 19, 1978. Histopathologic examination revealed an irregular corneal epithelium that was absent in many areas and hyperplastic in others. A degenerative pannus was present, and there was extensive stromal necrosis with vascularization and a dense infiltrate of inflammatory cells. The iris showed necrosis and atrophy of the stroma and almost total disappearance of the sphincter and dilator muscles. There were posterior synechiae. Rubeosis iridis, together with extensive peripheral anterior synechiae and hyphema, were noted. The only evidence of residual tumor cells was found in the inferior temporal quadrant where, within the longitudinal portion of the ciliary muscle, there was a group of tumor cells (Fig. 4) with large, irregularly shaped hyperchromatic nuclei, prominent nucleoli, and a vacuolated cytoplasm (Fig. 5). The lens was cataractous. Macrophages and acute inflammatory cells were present in the vitreous. The retina revealed loss of ganglion cells. The choroid was normal. The optic disk showed cupping and posterior bowing of the lamina cribrosa.

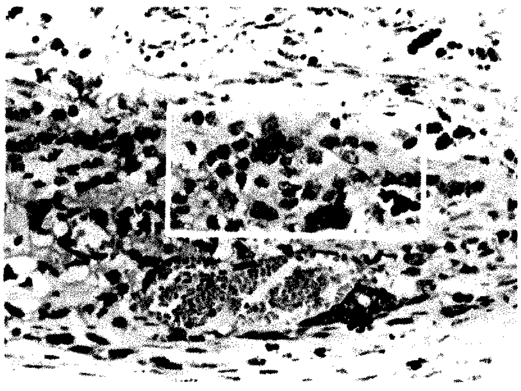

Fig. 4 (Frank and associates). Clumps of residual tumor cells in ciliary muscle after massive treatment by various modalities. The area enclosed by the white rectangle is enlarged in Figure 5 (hematoxylin and eosin, ×160).

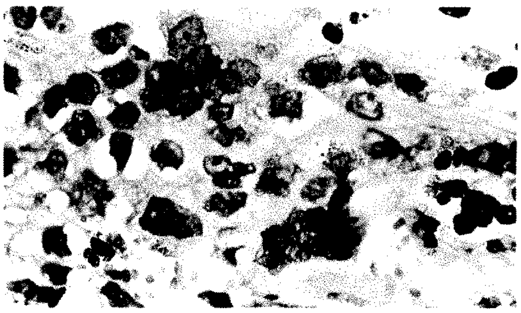

Fig. 5 (Frank and associates). Same tumor cells as shown in Figure 4 (within rectangle). Note vacuolization of cytoplasm and nuclei. (hematoxylin and eosin, ×630).

After enucleation the patient's condition continued to deteriorate. Human chorionic gonadotrophin levels rose to 360 mIU/ml. Subsequent courses of chemotherapy were cautiously given because of the development of jaundice, thought to be caused by drug toxicity rather than by the development of hepatic metastases. The patient died in January 1979. Permission for autopsy was not obtained.

DISCUSSION

It is unusual that ocular metastases occurred solely in the anterior segment. Choriocarcinomas metastasize via the bloodstream, and because there are no lymphatics in the eye, ocular metastases from this tumor should be located preferentially in the posterior uvea because of the larger volume of blood flow to the choroid than to any other region of the eye.

Histopathologic evidence showed that aggressive treatment by several modalities caused regression of this patient's ocular tumor but did not totally destroy it, and contributed to the complications that led to loss of the eye. The tumor cells present in the enucleated eye showed typical treatment changes that can be caused either by radiation or by chemotherapy, but one cannot conclude from the histologic presence of such changes that these cells are no longer viable. The corneal destruction represents complications of treatment,[24-27] whereas the abnormalities present in the iris and lens may be caused by the direct effects of treatment or by the persistent uveitis, which was perhaps exacerbated by the presence of material released by necrotic tumor cells.

A good prognosis for effective treatment of choriocarcinoma by chemotherapy generally depends on several factors: a relatively short duration of the disease before initiation of therapy, a pretreatment human chorionic gonadotrophin level that is not markedly increased, the absence of cerebral or hepatic metastases at the outset of treatment, and the presence of a gestational, rather than a nongestational, type of neoplasm.[8,28] In our case, the patient's clinical disease, dated from her last menstrual period, could not have been more than five months in duration, and she initially had no detectable cerebral or hepatic metastases, but her human chorionic gonadotrophin levels were massively increased. It is difficult to determine whether her tumor was of the gestational or of the nongestational type. Gestational choriocarcinomas primary in the ovary are extremely rare; however, nongestational pure primary ovarian choriocarcinomas unaccompanied by teratomatous elements are also unusual.[28] No products of conception could be found in the specimen obtained at laparotomy, but it is possible that this tumor may have represented an ovarian metastasis from a small and undetected uterine primary.

SUMMARY

A 28-year-old woman with ovarian choriocarcinoma developed a uveitis in her left eye. Subsequently, a mass was observed in the anterior segment of that eye together with a subconjunctival mass that appeared to extend from it. Biopsy of the subconjunctival nodule showed two different cell types consistent with the cytotrophoblastic and syncytiotrophoblastic elements typical of choriocarcinoma. No abnormalities of the posterior segment were found on careful examination. Treatment consisted of systemic chemotherapy with a variety of therapeutic agents, argon laser photocoagulation of tumor seedings in the anterior chamber angle, radiation to the eye, and finally, subconjunctival injections of methotrexate. The eye became blind and painful and was enucleated. Histopathologic examination revealed residual tumor cells in the anterior segment indicating treatment changes, but there were no abnormalities posteriorly. Choriocarcinoma metastatic to the eye has been reported infrequently, and this is the first case in which anterior segment metastases have been observed and the effects of treatment thoroughly documented.

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TREATMENT OF SYMPTOMATIC RETINAL BREAKS

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The purpose of prophylactic treatment of retinal detachment is to close the retinal breaks permanently. However, there is a considerable disparity between the incidence of retinal breaks in the general population and the estimated incidence of retinal detachment (1 in 10,000 to 20,000 individuals).[1-2]

The incidence of retinal breaks in the normal population determined by clinical examination has been from 5.8% in 1,700 patients reported by Byer[3] to 13.7% in 102 presumably normal subjects studied by Rutnin and Schepens.[4] Foos[5] showed 3.7% of retinal breaks in 2,406 eyes at autopsy. The incidence of retinal tears in the younger population was unknown, and for this reason we did a systematic examination of the retinal periphery in 150 boys, whose age averaged 14 years; retinal breaks were found in ten patients (6.6%) and a retinal dialysis in one (0.66%).[6] This study showed that the incidence of retinal breaks in the young is similar to the reported data in the general population and most of them will not become complicated by a retinal detachment.

Treatment should be reserved, therefore, to retinal breaks considered particularly dangerous. Treatment is generally advisable for breaks in aphakic eyes, in fellow eyes, and is clearly indicated for all symptomatic, fresh tears.

Fresh tears in patients with posterior vitreous detachments (photopsia and floaters), with or without vitreous hemorrhage, have a higher risk of detachment. Colyear and Pishel[7] reported on a group of 42 patients; in 12 patients (28.6%), retinal detachment followed. Davis[8] found 35% of retinal detachment in 31 eyes with symptomatic breaks on follow up.

We report herein the results obtained with the treatment of symptomatic retinal breaks including long-term follow up of 43 patients who were treated with cryopexy, and a comparable group of 43 patients who were treated with photocoagulation.

MATERIAL AND METHODS

One eye each of 43 patients was treated with photocoagulation. The average patient age was 58 years. Sixteen of the eyes were myopic by 2 diopters or more and two were aphakic. In the group of 43 eyes treated with cryopexy, the average patient age was 58 years; 14 eyes were myopic and one was aphakic.

All the patients had symptoms, either in the form of light flashes or floaters, from a recent vitreous collapse or had a vitreous hemorrhage. Patients with asymptomatic tears and breaks in fellow eyes were excluded (Table 1).

The type and location of the retinal breaks are summarized in Tables 2 and 3. Most are of the horseshoe type, followed by round holes with adherent or free opercula. Round holes without opercula were only seen associated with tears of the former types and were not considered the cause for the symptoms.

Of the 43 patients treated with photocoagulation, 13 had retinal disease in the fellow eye, shown before, at, or after the treatment of the retinal break; retinal de-

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TABLE 1

SYMPTOMS IN 96 EYES WITH RETINAL BREAKS

Symptoms	Eyes Treated with Photocoagulation	Eyes Treated with Cryopexy
Vitreous hemorrhage	16 (37.2%)	18 (41.8%)
Photopsia and floaters	27 (62.8%)	25 (58.2%)
Total	43	43

tachment was present in six and retinal breaks in seven patients. The same type of retinal disease was shown in the fellow eye of the group treated with cryopexy; retinal detachment in eight and breaks in six patients.

The duration of follow up ranged from 12 months to 12 years in the group treated with photocoagulation (average was 67 years, eight months). In the group treated with cryopexy, duration of follow up ranged from six months to eight years; average was 24.4 months).

RESULTS

Thiry-one patients in each group had no complications of any kind (72%) (Table 4). New tears without retinal detachment was the most frequent posttreatment complication, and occurred in six (14%) of the cases in both groups. In the eyes treated with photocoagulation, the new tears were recognized about one month after treatment in four of six cases; the patients complained of floaters. The new tears were of the horseshoe type; small in size in most cases, and tended to form in the opposite hemisphere (Fig. 1).

In the eyes treated with cryopexy, the new tears were recognized a few months after treatment; all were of the horseshoe type and were located randomly. The original break was of the horseshoe type in five out of six cases, in both groups. New tears were treated with the method used for the original breaks, without further complications. Initial visual acuity was maintained.

Three patients (7%) developed symptoms related to intravitreal hemorrhage after photocoagulation. The hemorrhage originated in an avulsed retinal vessel that bridged a horseshoe flap. A patient with recurrent intravitreal hemorrhage required a scleral buckling operation to counteract the vitreous traction over the flap and vessel.

The overall incidence of retinal detach-

TABLE 2

TYPE AND DISTRIBUTION OF BREAKS IN 43 EYES TREATED WITH PHOTOCOAGULATION

Type of Break	Quadrant: Superior Temporal	Superior Nasal	Inferior Temporal	Inferior Nasal
Horseshoe tear	16	19		
Holes with opercula	8	5	1	
Round holes without opercula	1	1		
Others	3*			

*Two elongated, perivascular holes and one giant retinal tear (160 degrees), without detachment.

TABLE 3

TYPE AND DISTRIBUTION OF BREAKS IN 43 EYES TREATED WITH CRYOPEXY

	Quadrant			
Type of Break	Superior Temporal	Superior Nasal	Inferior Temporal	Inferior Nasal
Horseshoe tear	25	7	3	4
Holes with opercula	6	2	2	2
Round holes without opercula	2	2		
Others	1*			

*Elongated break along posterior border of area of lattice degeneration.

TABLE 4

POSTTREATMENT COMPLICATIONS IN 96 TREATED EYES

Complications	Patients Treated with Photocoagulation	Patients Treated with Cryopexy
New tears without detachment	6* (14%)	6* (14%)
Recurrent vitreous hemorrhage	3	0
Retinal detachment from treated areas	1†	3†
Retinal detachment from untreated areas	2†	2†

*The original break was of the horseshoe type in five of the six cases in both groups.
†The original break was of the horseshoe type in all cases that became detached.

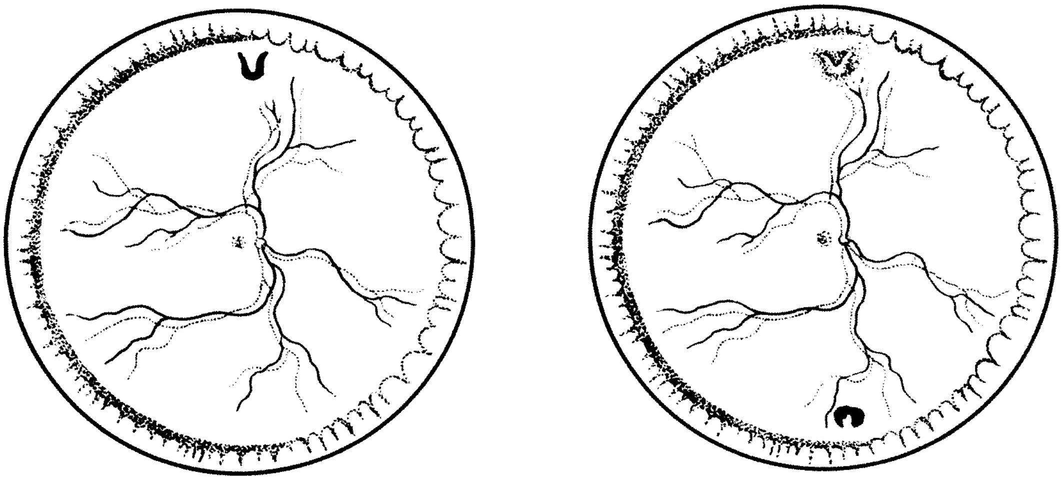

Fig. 1 (Verdaguer and Vaisman). New tears without retinal detachment. Left, a fresh superior horseshoe tear was treated with photocoagulation. Right, One month later, the patient had a new episode of floaters and a new tear was found inferiorly.

ment during the observation interval was 7% (three patients) for the group treated with photocoagulation and 11.6% (five patients) for the eyes treated with cryopexy. The original break was of the horseshoe type in all patients. Two eyes developed retinal detachments arising from new tears, seven years and four months, respectively, after photocoagulation. A successful scleral buckling was performed in both cases. (Fig. 2). Two eyes developed retinal detachment with new breaks after three months, in one case, and 19 months of cryopexy. One of these retinas was successfully reattached surgically; the other eye developed massive vitreous retraction and preretinal membrane formation. One patient treated with photocoagulation had a retinal detachment and a tear located at the border of the treatment scar. The retina was successfully reattached surgically, but visual acuity was reduced to 6/60 (20/200) because of myopic macular changes. Three patients with cryopexy developed detachment in the region of the original break. Retinal detachment occurred one month (Fig. 3), six months, and seven years after treatment, respectively. Two of these patients were successfully operated on and one developed massive vitreous retraction and preretinal organization.

Only one patient treated with cryopexy developed a cellophane maculopathy with cystoid macular edema.

DISCUSSION

We treated patients with retinal breaks who had a high risk of retinal detachment, with symptoms of vitreous traction and vitreous hemorrhage in most eyes. Studies on the course of retinal breaks suggest that 30 to 50% of eyes with fresh horseshoe tears will develop detachment if untreated.

Our study included one eye treated with photocoagulation and another treated with cryopexy that developed retinal detachment arising from new tears years after the initial procedure. These probably should not be considered as failures of the treatment. Our results are similar to those of Robertson and Norton,[9] who reported 6% retinal detachment in 301

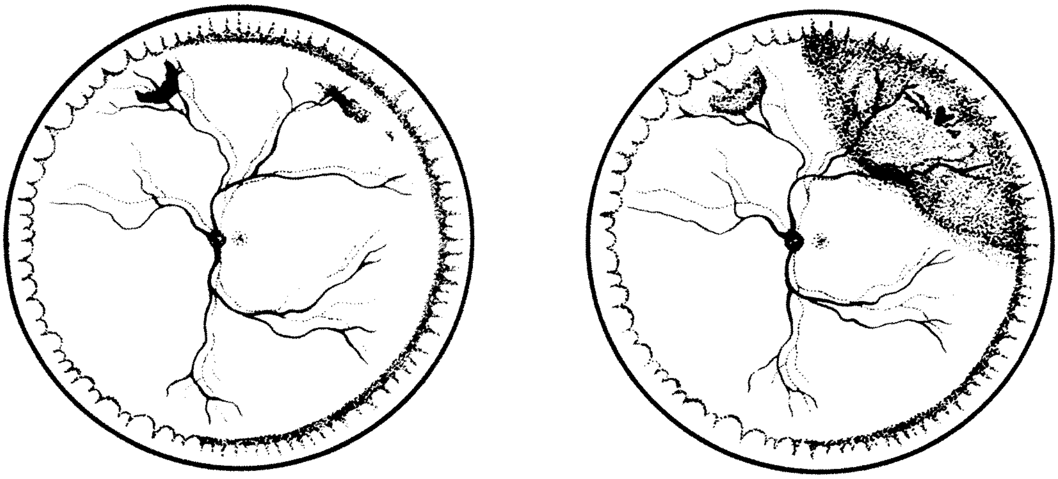

Fig. 2 (Verdaguer and Vaisman). Retinal detachment arising from new tears. Left, A superiornasal horseshoe tear was treated with photocoagulation. Right, The patient had a retinal detachment four months later with a new break in an area of lattice degeneration.

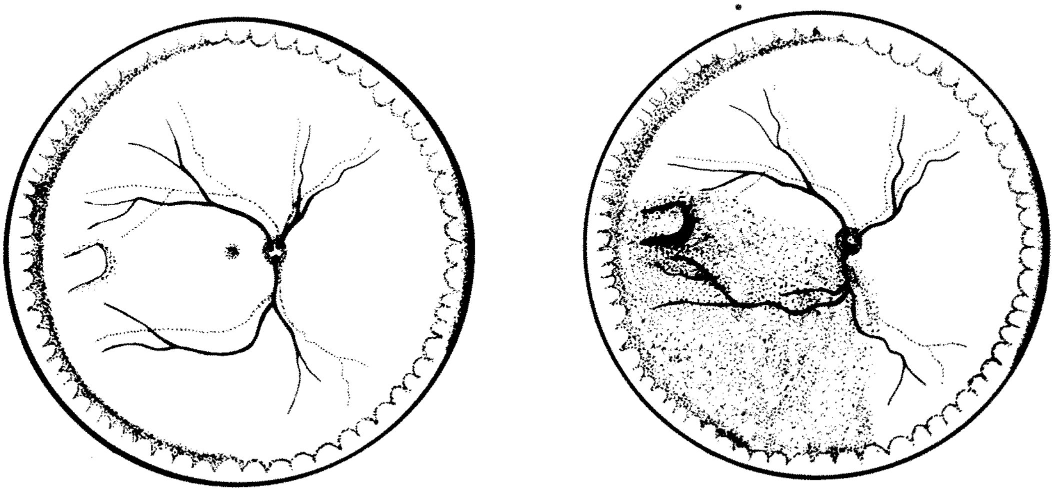

Fig. 3 (Verdaguer and Vaisman). Retinal detachment arising from treated area. A patient treated with cryopexy for a horseshoe tear (left) developed a retinal detachment arising from the original break (right).

treated eyes, but their study included different varieties of retinal breaks, including asymptomatic tears.

In our series the original retinal break was a horseshoe-type tear in all cases, which emphasizes the importance of vitreous tractions in these eyes in which cryocoagulation or photocoagulation failed to prevent retinal detachment. The detachment developed in the region of the original break in three patients treated with cryopexy; this may suggest that the cryocoagulation scar is not strong enough in some cases.

Cryopexy is preferred by many ophthalmologists for the treatment of retinal breaks; our study, however, does not show the superiority of this method over photocoagulation. Because cryopexy is easier to perform, we believe it should be used for breaks anterior to the equator. Photocoagulation is preferable for more posteriorly placed lesions.

SUMMARY

Forty-three patients with symptomatic retinal breaks were treated with photocoagulation and a comparable group of 43 patients were treated with cryopexy. All patients had either a vitreous hemorrhage, or photopsia and floaters. Most of the breaks were of the horseshoe type. Of the patient eyes, 14% (six patients) in both groups developed new retinal tears without detachment during the long term postoperative observation. The new breaks were treated with the method used for the original breaks, and no scleral surgery was necessary.

The incidence of retinal detachment was 7% in patients treated with photocoagulation and 11.6% in patients with cryocoagulation, but two cases that developed retinal detachment arising from new tears years after the initial procedure were included.

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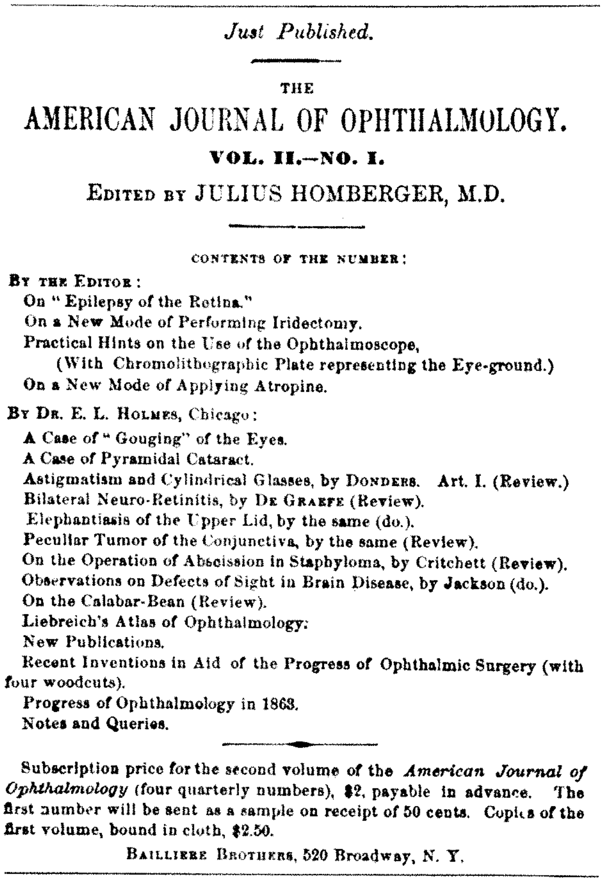

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VOL. II.—NO. I.

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BAILLIERE BROTHERS, 520 Broadway, N. Y.

From American Medical Times Advertiser
April 2, 1864

THE AUTOSOMAL DOMINANT SYNDROME OF PROGRESSIVE OPTIC ATROPHY AND CONGENITAL DEAFNESS

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Konigsmark and associates[1] described six persons in four generations of a family with a dominantly inherited association between congenital deafness and progressive optic atrophy. We describe herein a second family affected by congenital deafness and progressive optic atrophy (Figure). Hearing evaluation revealed that the family members with this syndrome had a potentially treatable form of deafness. The affected individuals could hear amplified speech, even though they could not recognize and repeat the words because they had learned only manual language. If they had had amplification in early childhood and training in oral as well as in manual language, they could have developed adequate speech.[2,3]

CASE REPORTS

Case 1—The proposita, a 48-year-old deaf woman (IV-2, Figure), noted bilateral gradual decrease in vision beginning at age 30 years. Bilateral optic disk pallor and severe deafness were the only neurologic abnormalities. Visual acuity was corrected to R.E.: 6/30 (20/100) and L.E.: 6/21 (20/70) with -1.50 sphere. An enlarged blind spot and a central scotoma were plotted by Goldmann perimetry in the visual field of each eye. The patient's pupils reacted normally. Bilateral eyelid signs of Graves' disease were present. Results of the slit-lamp examination and intraocular pressures were normal. Ophthalmoscopic examination showed moderate pallor of the optic disk in each eye. Vessel caliber, posterior pole, and peripheral retina were normal. Results of electroretinography and electro-oculography were normal for the patient's age.

Thyroid studies confirmed the diagnosis of Graves' disease, which required radioiodine therapy. Follow-up visits six months and one year later revealed resolution of eyelid signs of Graves' disease. No deterioration of vision occurred during this follow-up period.

Case 2—The 25-year-old son (V-1, Figure) of the proposita had been deaf since birth. He had complained of slight decrease in vision in both eyes for the past three to four years. On examination, his best corrected visual acuity was R.E.: 6/9 (20/30) and L.E.: 6/12 (20/40) with -1.00 sphere. Results of external and slit-lamp examinations were normal. Results of the ophthalmoscopic examination were normal except for a generalized slight pallor of both optic disks. The right disk had a 0.4 cup-disk ratio, and the left disk had a 0.5 cup-disk ratio. Visual field examination showed no central scotoma or enlargement of the blind spot. The electroretinogram was normal.

Cases 3 and 4—The 23 and 24-year-old sons of the proposita had normal visual acuity and normal optic disks. The 24-year-old son (V-2) had normal hearing, whereas the 23-year-old son (V-3) was congenitally deaf.

Cases 5 and 6—The proposita's first cousin (III-2) and her maternal aunt (IV-1) had bilateral optic atrophy and congenital deafness. The mother was 70 years old at the time and the daughter was 47 years old. The daughter had visual acuity of 6/30 (20/100) in each eye with a refractive error of -3.0 sphere.

The proposita's deceased maternal uncle and aunt (III-3 and III-4) and her maternal grandmother (II-6) had congenital deafness and moderately poor vision occurring in the third or fourth decade of life. However, visual loss did not progress to complete blindness.

Four deaf members of this family, the proposita, her husband, and her two sons (IV-2, IV-3, V-1, and V-3), were given hearing evaluations. Pure tone audiometry and speech reception thresholds were conducted in a double wall sound-treated room with a one-way window. Earphones with cushions were used for all tests administered through an audiometer. The thresholds of hearing sensitivity were bilaterally symmetrical in the proposita and her two sons, sloping from a severe impairment in the lowest test frequencies to profound deafness at 1,000 Hz. Speech reception thresholds were defined as the level at which spondaic words could be heard. Because these three patients used manual language and had little oral speech, it was impossible for them

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This study was supported in part by the Ohio Lions Eye Research Foundation.

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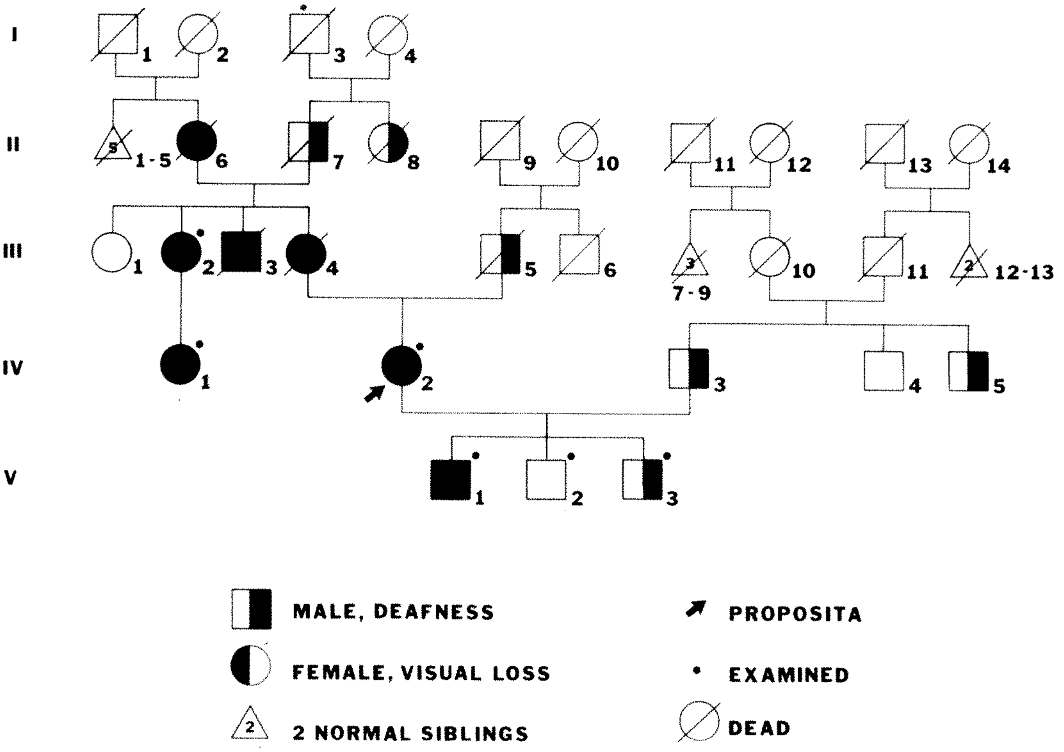

Figure (Kollarits and associates). Pedigree showing seven affected persons in the last four generations of this family.

to repeat words. The father in this family was also deaf. He was unable to hear either pure tones or speech at the maximum output of the audiometer. The pure tone thresholds of the two deaf sons resembled the audiogram of their mother.

DISCUSSION

The five known hereditary syndromes[4] in which hearing loss is associated with optic atrophy include: progressive optic atrophy and congenital sensorineural deafness[1]; optic atrophy with diabetes and sensorineural hearing loss[5]; optic atrophy, ataxia, and progressive sensorineural hearing loss (Sylvester syndrome)[6]; opticochleodentate degeneration[7]; and optic atrophy, polyneuropathy and sensorineural deafness.[8] The mode of inheritance and associated characteristics of patients with these syndromes are listed in the Table.

This family had the syndrome previously described as dominantly inherited progressive optic atrophy and congenital sensorineural deafness.[1] With the exception of the Sylvester syndrome, the mode of inheritance in the other syndromes with hereditary deafness and optic atrophy is autosomal recessive or X-chromosome linked recessive.[5,7,8] The Sylvester syndrome (optic atrophy, ataxia, and progressive sensorineural hearing loss) has a dominant mode of inheritance, but none of the members of our family had early onset of visual loss or ataxia, as seen in Sylvester syndrome.[6] The age at which visual loss occurs in patients with progressive optic atrophy and congenital sensorineural deafness was reported previously as being about age 30 years.[1] Our patients noticed a decrease in vision between age 24 and 30 years.

Age of onset and severity of hearing loss also distinguish progressive optic atrophy and congenital sensorineural deaf-

TABLE

SYNDROMES WITH HEREDITARY ASSOCIATION OF OPTIC ATROPHY AND DEAFNESS

Syndrome	Inheritance	Vision	Hearing	Associated Findings
Progressive optic atrophy and congenital sensorineural deafness[1,3]	Autosomal dominant	Progressive optic atrophy with visual loss in middle to late life	Congenital severe sensorineural deafness	None
Optic atrophy with diabetes mellitus and sensorineural hearing loss[3,5]	Autosomal recessive	Progressive visual loss Bilateral optic atrophy before age 16 years	Onset in childhood. Progressive hearing loss	Diabetes mellitus with onset in the first or second decade
Optic atrophy, ataxia, and progressive hearing loss (Sylvester syndrome)[3,6]	Autosomal dominant	Progressive visual loss secondary to optic atrophy onset 2 ½ to 9 years of age	Moderate to severe progressive sensorineural deafness	Ataxia particularly involving the legs, weakness and muscle wasting particularly involving the shoulder girdle and hands
Opticocochleodentate degeneration[3,7]	Autosomal recessive	Optic atrophy with onset of visual loss in infancy	Progressive hearing loss resulting in severe deafness	Onset in infancy of progressive spastic quadriplegia, progressive mental deterioration, and death in childhood
Optic atrophy, polyneuropathy, and sensorineural deafness (Rosenberg-Chutorran syndrome)[3,8]	X-chromosome linked or autosomal recessive	Visual loss with optic atrophy beginning at age 20 years	Progressive sensorineural hearing loss with deafness by age 6 years	Progressive peripheral polyneuropathy beginning in early childhood

ness from the other four syndromes. In this family, the hearing loss was congenital and severe. Konigsmark and associates[1] noted the same finding. This is in contrast to the other four syndromes. In all of these, the hearing loss was progressive with onset either in infancy or childhood. Of the five syndromes with hereditary optic atrophy and sensorineural hearing loss, the one that this family had is the only one without associated anomalies or systemic illness. The other syndromes are associated with various disorders including diabetes mellitus, ataxia, progressive spastic quadriplegia and mental deterioration, or progressive peripheral polyneuropathy.[4] None of the members of this family showed evidence of having any of these disorders. One family member in this pedigree did have Graves' disease that was treated medically.

SUMMARY

Four members of a family had the heriditary syndrome of dominantly inherited progressive optic atrophy and congenital sensorineural deafness. Hearing evaluations revealed that two members had a potentially treatable form of deafness.

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RHEGMATOGENOUS RETINAL DETACHMENT COMPLICATING CYTOMEGALOVIRUS RETINITIS

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Cytomegalovirus is an ubiquitous DNA virus of the herpesvirus group. Complement fixation antibodies to this agent have been found in 81% of the adult population over age 35 years.[1] Clinical manifestations of the virus, however, are almost entirely limited to neonates and adults with leukemia, lymphoma, or iatrogenic suppression of the immune system after organ transplantation.[2] Involvement of the eye occurs primarily as a necrotizing retinitis with a variable degree of associated uveitis.[3-9] Optic nerve involvement,[4,9,10] and isolated anterior uveitis have also been reported.[11] Virus has been isolated from subretinal fluid,[5] aqueous humor,[10,11] and tears.[12]

Cytomegalovirus retinitis has been reported to be responsive to transfer factor,[13] but other attempts at therapy have been disappointing. Retinal detachment has been reported as a complication in only two patients.[5,14] We report herein an expansion on the features of one of these cases[5] (Case 1) and report the clinical appearance, course, and management of retinal breaks and rhegmatogenous detachment in a total of six eyes in four patients.

CASE REPORTS

Case 1—A 23-year-old man was first seen on April 27, 1970, because of recent decrease in vision. In September 1969, he had undergone renal transplantation. On examination, visual acuity was R.E.: 6/7.5 (20/25) and L.E.: 6/6 (20/20). Examination of the right eye revealed sheathing of the superotemporal vein with yellow material, beginning one disk diameter away from the disk and extending into the temporal periphery. The left eye also showed sheathed vessels and exudate. Urine was positive for cytomegalovirus. The retinitis abated with reduction of systemic corticosteroid dosages. In March 1971, a serous exudative detachment developed in the right eye, and it remained constant for six months. In May 1972, there was sudden loss of visual acuity in the left eye to counting fingers. A vitritis was noted and the superior 60% of the retina was detached. Two holes were present between the ora serrata and equator, and multiple thin areas were noted in the superior retina. Extensive periretinal proliferation was present. Surgery was performed with cryotherapy and encircling silicone exoplants. Cytomegalovirus was cultured from the subretinal fluid.[5]

Five months later, fluid was leaking over the buckle at the 5 o'clock position and xenon photocoagulation was applied over the entire superotemporal region, which resulted in re-attachment. At this time the right eye developed detachment with a hole at the ora serrata and equatorial holes. Surgical management with a localized sponge exoplant was successful in reattaching the retina. Cytomegalovirus was also cultured from the subretinal fluid in this eye.

In February 1973, a recurrent detachment developed in the left eye caused by formation of new holes posterior to the buckle, and repeat scleral buckling was performed. New areas of active retinitis developed in the right eye in December 1973. On March 13, 1974, therapy with transfer factor was begun. Two weeks later there was a gradual reduction of inflammation and by May 23, 1974, all the lesions in the right eye became inactive. Urine cytomegalovirus titers dropped from $10^{3.5}$ $TCID^{50}$ to $10^{0.5}$ during transfer factor therapy.[13]

Because of the continued inferior traction detachment in the left eye, a total posterior vitrectomy and lensectomy were performed in the left eye in September 1974. Postoperatively the patient continued to have total retinal detachment posterior to the buckle, secondary to persistent traction. Visual acuity remained 6/7.5 (20/25) in the right eye and hand motions in the left eye.

Case 2—A 52-year-old woman was first seen on Feb. 7, 1974. She complained of flashing lights in the left eye of two weeks' duration and black spots of five days' duration. She had undergone a renal

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transplant in 1972 and subsequently developed cataracts that were removed without complication. Examination of the left eye revealed a vitreous hemorrhage and a retinal tear approximately one-half disk diameter in size at the 6:30 position and 5 to 6 disk diameters from the disk. The configuration was a reverse horseshoe pointing toward the ora serrata. Posterior to this area, shaggy, necrotic areas of retina with multiple partial thickness holes were noted. On Feb. 8, 1974, the two rows of cryotherapy were placed around the hole. Cultures of urine were positive for cytomegalovirus. Cryotherapy lesions healed normally and visual acuity remained 6/6 (20/20). In November 1974, she complained of floaters in the previously unaffected right eye. There was vitreous haze and a large, apparently necrotic area surrounded by hemorrhage at the 11 o'clock position near the equator. Five days later, vitreous haze had increased and a large hole with shaggy edges had developed in this area accompanied by a superotemporal detachment. A retinal re-attachment procedure was performed with cryotherapy and encirclement with external soft silicone elements. Postoperatively the hole appeared closed but subretinal fluid continued to increase. Repeat scleral buckling was performed two weeks later. On examination two months later, visual acuity was counting fingers at 4 feet. A high buckle was present and no open holes could be identified, although some residual superotemporal fluid remained. The patient was subsequently lost to follow-up.

Case 3—A 50-year-old man was first seen on Feb. 18, 1975, complaining of decreased visual acuity in the right eye with spots and threads of one day's duration. He had renal transplantation in May 1973. Visual acuity was 6/6 (20/20) in each eye. In the right eye, white areas were collected in patches in the retina. Superotemporally there was a 5- to 7-disk diameter area of gray appearing retina that was interpreted as retinal necrosis with hole formation accompanied by intraretinal hemorrhages. No retinitis in the left eye was noted. Cytomegalovirus titers by complement fixation were 1/128. Cryotherapy was applied around the hole with formation of an apparent adequate adhesion. Over the next two months, he maintained 1 to 2+ flare and cell in the anterior chamber but there was no significant change in the retinal findings. On Oct. 1, 1975, visual acuity decreased in the right eye to counting fingers and extensive massive periretinal proliferation was noted with a new open hole and detachment. The patient refused surgery and died of a myocardial infarction one week later.

Case 4—A 39-year-old man underwent renal transplantation in May 1970 with repeat transplantation in February 1971. He had noted decreased vision in the right eye since August 1970 and when first examined in November 1971, visual acuity was R.E.: hand motions and L.E.: 6/7.5 (20/25). The vitreous showed heavy discrete exudates and there were perivascular, subretinal, and intraretinal exudates characteristic of cytomegalovirus retinitis. Examination of the fundus was difficult because of the heavy vitreous haze. Corticosteroid dosage was decreased and the retina became more visible. Posterior synechiae developed secondary to new anterior segment inflammation and visual acuity in the right eye decreased to light perception three months later. The vitreous slowly cleared over the next three months to reveal a total rhegmatogenous retinal detachment that was inoperable because of massive periretinal proliferation.

DISCUSSION

Cytomegalovirus retinitis is typically manifested as a marked perivascular exudative and hemorrhagic response. The differential diagnosis must include disseminated toxoplasmosis and herpes simplex retinitis. The appearance of this in an adult who has a lymphoproliferative disorder or who has been treated with immunosuppressive agents, coupled with isolation of the virus from urine, saliva, tears, or sputum establish the diagnosis. The disease typically progresses peripherally along vessels with time. The retinitis may resolve spontaneously in some instances or may respond to reduction of immunosuppressives.[5,13] Transfer factor has caused inactivation of clinical disease and simultaneous marked reduction of urine excretion of the virus in one case.[13]

Exudative retinal detachment may complicate cytomegalovirus retinitis. Retinal tears, however, appear in necrotic areas and may be difficult to recognize because of vitreous haze, a uniform finding. The differential diagnosis between exudative detachment and rhegmatogenous detachment may be therefore difficult. Systemic corticosteroids should not be increased to reduce vitreous reaction because this may actually worsen the condition. Reduction of systemic corticosteroids and other immunosuppressive agents, addition of topical corticosteroids and dilatation to treat anterior uveitis, and therapy with transfer factor[13] should be considered. Follow up with serial ultrasonography may be valuable if vitreous haze is so severe and persistent that it prevents adequate examination of the fundus.

When retinal holes can be identified as in Cases 1 to 3, they are usually large, round with shaggy edges, and are located in areas of retinal necrosis. In our cases, they were present anywhere from far posterior (5 disk diameters from the disk) to the ora serrata. One hole had the appearance of a reversed horseshoe tear pointing toward the ora serrata. Holes are frequently multiple; recognition of complete breaks is made difficult not only by vitreous haze but by the association of multiple thin atrophic areas. Simple holes may respond to cryotherapy alone, at least initially as in Cases 2 and 3. Detachment may be managed successfully with cryotherapy and exoplant surgery, but interpretation of the postoperative course can be difficult because residual fluid may be seen more frequently than usual as the result of exudative reactions, requiring differentiation from continued leakage caused by open holes as in Cases 1 and 2.

Continuing difficulty may occur in these eyes because of the appearance of new holes, even as much as 28 months after initial surgery (Case 1). New areas of retinitis may also develop after successful buckling surgery as in Case 1.

Massive periretinal proliferation is a common occurrence and was found in three of the six eyes in our cases. In one eye it was noted with recurrent hole formation six months after successful cryotherapy and was accompanied by new hole formation. A second eye had such extensive proliferation that when the fundus was visualized after clearing of the vitreous haze, it was considered to be inoperable. The third eye had significant massive periretinal proliferation present at the time of initial detachment but was successfully repaired. After redetachment nine months later, however, conventional retinal surgery followed by vitrectomy as a third procedure failed to re-attach the retina because of the presence of massive periretinal proliferation.

With continued follow up, formation of holes and detachment involved the second eye five months (Case 1) and nine months (Case 2) after surgical therapy to the first eye. One patient died one week after detachment developed in the first eye. The occurrence of late redetachment and new retinitis in initially successfully operated eyes and a tendency for detachment to occur in the second eye make continued long-term careful follow up an important facet of management of these patients.

SUMMARY

Six eyes in four patients with cytomegalovirus retinitis developed retinal holes and retinal detachment. Holes appeared in areas of necrosis and were typically large and round with shaggy edges. Differentiation from exudative detachment was sometimes difficult because of obscuration of the fundus by vitreous haze. Cryotherapy was successful in closing open breaks without detachment; cryotherapy and exoplant surgery were used effectively to treat the initial detachments. The occurrence of massive periretinal proliferation and late development of new areas of hole formation or retinitis complicated therapy for these detachments.

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RECONSTRUCTION OF THE MEDIAL EYELID

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When a full-thickness lower eyelid defect cannot be closed with a canthotomy and cantholysis, a temporal rotational flap[1,2] or eyelid sharing procedure[3] from the upper eyelid is usually considered. This is particularly true in the medial one third of the eyelid where there is little stretch of the medial canthal tissue. We describe herein a method of repairing medial defects of the lower eyelid by using a free tarsal-conjunctival graft and a nasal-based skin-muscle pedicle flap.

Material and methods

This procedure was usually done under local anesthesia using 2% lidocaine with hyaluronidase and epinephrine 1:100,000. The tumor was examined with the operating microscope and a clear 1.5-mm margin was drawn around the tumor (Fig. 1). The punctum or canaliculus were removed if necessary and externalization of the canaliculus was attempted only if a small portion of the punctum or canaliculus had been resected. The specimen was sent to the pathologist for a frozen section examination, and when free margins were reported, reconstruction was begun.

The upper eyelid was everted over a Desmarres retractor and a free tarsal-conjunctival graft 4 mm wide and corresponding in length to the size of the defect was removed. The graft was taken from the midportion of the tarsus, leaving at least 3 mm of tarsal support for the eyelid margin. The tarsal defect was left unsutured.

If the canaliculus was resected, the graft was sutured to the lower arm of the canthal tendon with a double arm suture of 4-0 Dacron. Otherwise, it was sutured to the tarsus of the remaining eyelid with 6-0 chromic catgut with the superior edge level with the eyelid margin. The graft was also sutured to the conjunctiva in the inferior cul de sac as well. The sutures were placed in such a way that the knots were tied on the anterior surface to avoid rubbing against the cornea. If the graft was too tight against the globe it was trimmed to avoid a postoperative sag.

The skin beyond the medial aspect of the defect formed the base of the pedicle flap that rotated into the defect. An outline of the flap was drawn and was slightly larger than the defect it would fill. The flap including orbicularis was cut, lifted from its bed, and rotated upward to cover the tarsal-conjunctival graft and the remainder of the defect. The skin was sutured to the tarsal-conjunctival graft with 6-0 silk to create the new eyelid margin, and the lateral aspect of the flap was sutured to the lateral eyelid segment. A full-thickness postauricular graft was placed in the defect created by the transfer of the pedicle flap to the eyelid margin.

A pattern of the defect was made with sterile paper, then traced in the postauricular area. The skin was injected with 2% lidocaine with epinephrine 1:100,000 and the area outlined was cut with a No. 15 Bard-Parker blade. The graft was removed from its bed by sharp dissection, leaving as much subcutaneous tissue as possible. The graft was placed in saline

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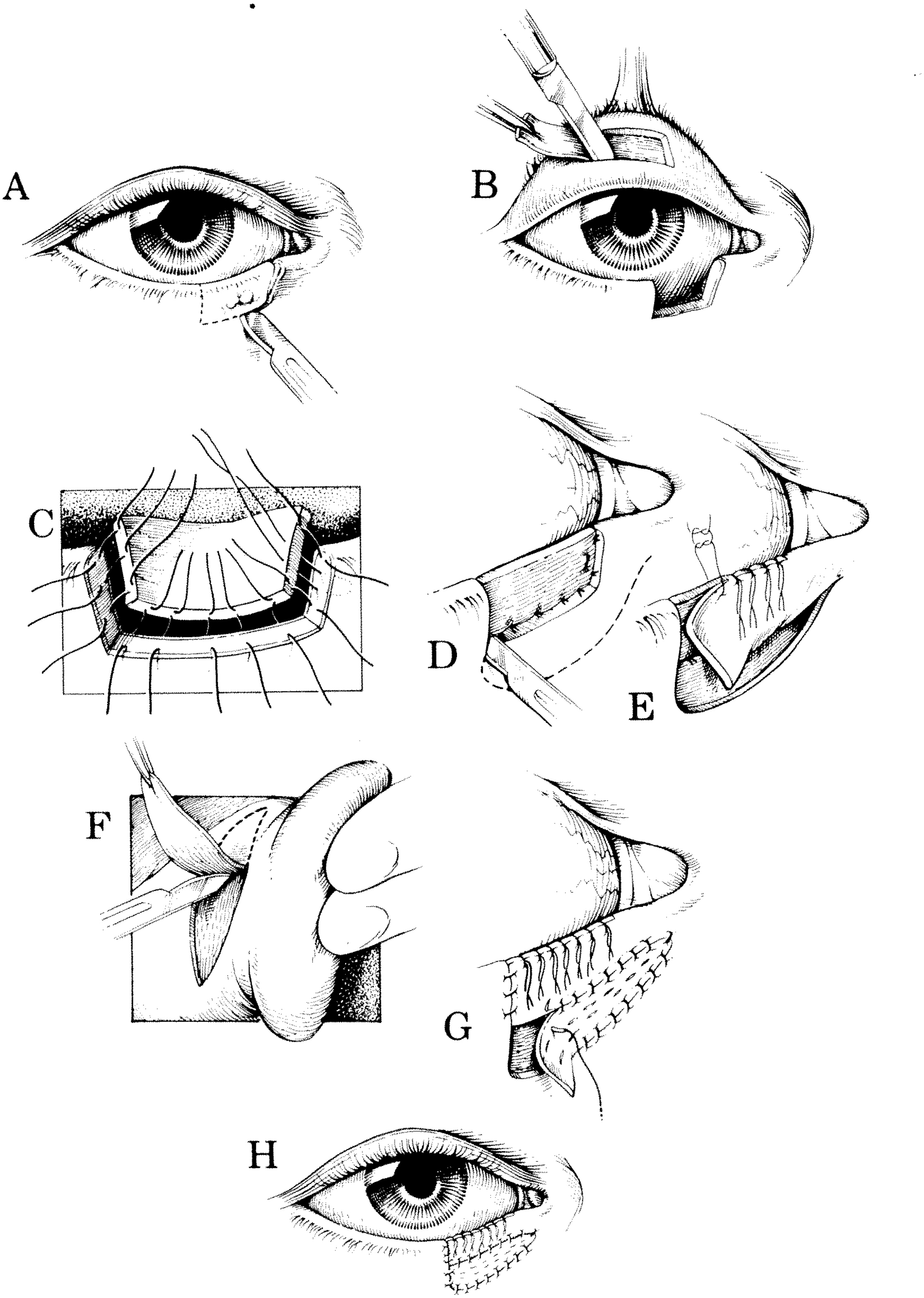
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while the closure of the defect was done with a continuous suture of 4-0 Prolene. Small stab incisions were made in the graft to allow for drainage. The graft was made to fit the defect with only a slight redundancy present. The graft was placed with 6-0 silk with the ends of the suture left long. At the superior edge where the graft was joined to the pedicle graft, suture bites were taken in the subcutaneous tissue to secure both edges to the deeper layer. A cotton roll soaked in neomycin sulfate (Neosporin drops) was placed over the graft and the silk sutures were tied over the stent as a pressure bandage. Antibiotic ointment was placed over the eyelid area and a wet loose dressing was applied. The sutures were removed in seven days.

CASE REPORTS

Case 1—A 49-year-old woman had a 6-mm recurrent basal cell carcinoma just lateral to the punctum in the right lower eyelid (Fig. 2). Using frozen section control, a 9-mm resection was carried out sparing the punctum. We used a free tarsal-conjunctival graft from the ipsilateral upper eyelid to fill the defect in addition to using a nasal-based skin-muscle pedicle flap. The secondary defect was repaired with a full thickness skin graft. Postoperatively, the patient has a cosmetically satisfactory appearance with a normal upward contour of the medial aspect of the lower eyelid.

Case 2—A 42-year-old man had a 15-mm resection of the medial aspect of his left lower eyelid including the punctum and canaliculus for basal cell carcinoma (Fig. 3). A 10-mm tarsal-conjunctival graft was taken from the ipsilateral upper eyelid to fill the defect. Because of tension on the suture line between the graft and eyelid, it was necessary to do a small canthotomy and cantholysis at the lateral canthus. A nasal-based skin-muscle pedicle flap was brought from below to cover the tarsal-conjunctival graft, and the secondary defect was repaired with a full-thickness skin graft. One year postoperatively, he has a cosmetically satisfactory result with only occasional epiphora.

DISCUSSION

Defects of the medial aspect of the lower eyelid are difficult to repair as compared to the lateral canthal and temporal areas because of a lack of available tissue in the medial canthus. If a temporal rotational flap[1,2] is used to repair a moderately large defect, normal eyelid must be rotated nasally leaving an area laterally of unsupported tissue against the globe. This occasionally requires additional reconstruction or mucous membrane covering of the lateral eyelid area. A modified Hughes [3] procedure may be done but this requires occluding the fissure for several weeks until the upper eyelid can be released. Confining the reconstruction to the local area is preferable, and does not violate the lateral canthal area to any great extent. Thus, the risk of a cosmetic deformity in the uninvolved area can be avoided. However, a small canthotomy and cantholysis does not usually distort the lateral canthus, and as in Case 2, is sometimes necessary to relieve tension on wound edges.

The punctum and canaliculus may be removed to get tumor-free edges. If the distal aspect of the canaliculus is intact, we attempt to externalize it by leaving a

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Fig. 1 (Leone and Hand). A, The tumor is outlined and removed with a clear margin of at least 1.5 mm. B, The upper eyelid is everted and a free tarsal-conjunctival graft 4-mm wide and similar in length to the defect in the lower eyelid is removed leaving at least 3 mm of tarsus at the eyelid margin. This defect is left unsutured. C, The graft is sutured in place with interrupted sutures of 6-0 chromic catgut. If the graft is not fairly tight against the globe it should be trimmed. D, The skin and orbicularis below the defect form a pedicle flap slightly larger than the defect and is based in the medial canthal area. E, The skin and tarsal-conjunctival grafts are joined with interrupted sutures of 6-0 silk to form the new eyelid margin. Some undermining may be necessary to obliterate tractions lines. F, An ellipse of full thickness postauricular skin is removed and the donor site is closed with a continuous 4-0 prolene suture. G, In the defect created by the transfer of the skin pedicle flap, the full thickness postauricular graft is placed and sutured with interrupted 6-0 silk. Stab incisions are made in the graft for drainage. H, This is the immediate postoperative appearance.

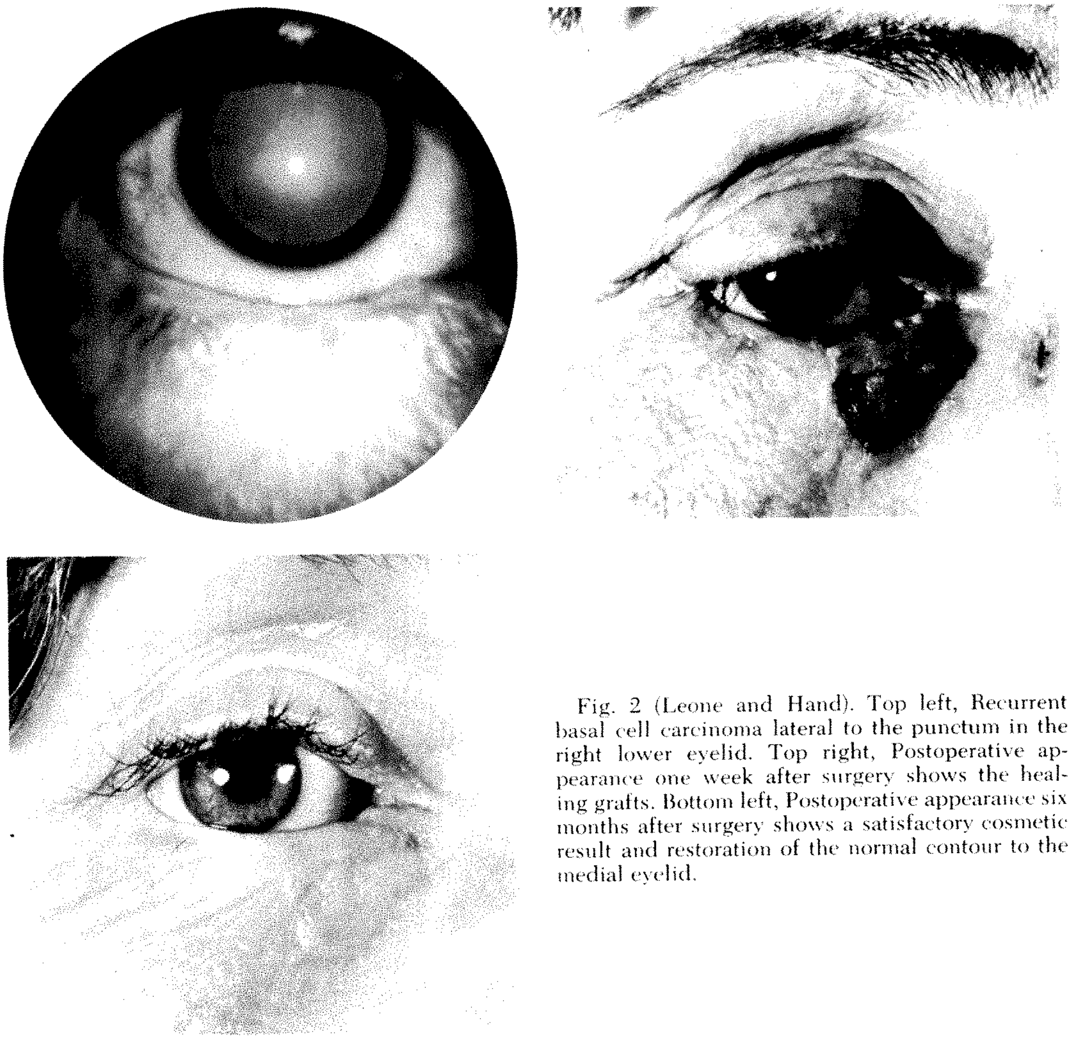

Fig. 2 (Leone and Hand). Top left, Recurrent basal cell carcinoma lateral to the punctum in the right lower eyelid. Top right, Postoperative appearance one week after surgery shows the healing grafts. Bottom left, Postoperative appearance six months after surgery shows a satisfactory cosmetic result and restoration of the normal contour to the medial eyelid.

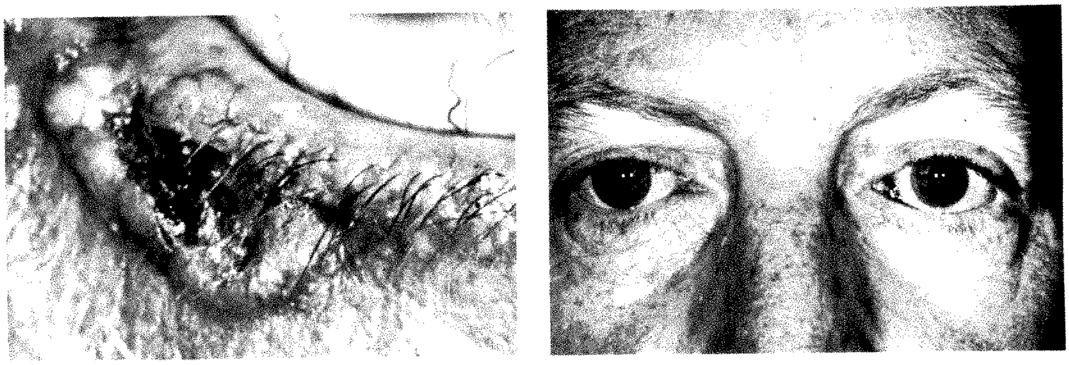

Fig. 3 (Leone and Hand). Left, Basal cell carcinoma occupying the medial aspect of left lower eyelid. Right, Postoperative appearance one year later after a 15-mm resection of the medial aspect of the left lower eyelid.

silicone tube within the orifice for four to six weeks while it is healing. However, if only a small portion of the proximal canaliculus is left, and if the upper canaliculus is functioning, no attempt is made to externalize it because it may interferre with the placement of the tarsal-conjunctival graft.

Because a newly reconstructed eyelid needs support, a tarsal-conjunctival graft is the ideal material. This is placed in the defect to simulate the tarsus that has been removed, and if there is no medial eyelid remaining, it is sutured to the remnant of the medial canthal tendon to retain the upward curvature of the lower eyelid and close apposition to the globe. The pedicle skin-muscle graft is necessary to nourish the free tarsal-conjunctival graft, and can be fitted in the area with a minimum of traction lines. Orbicularis is a necessary part of the pedicle flap because it is vascular and increases the blood supply brought to the tarsal-conjunctival graft. In the secondary defect, a postauricular skin graft is preferred to upper eyelid skin because the shrinkage factor is less. The advantage to this procedure is that the lateral canthus is left undisturbed and the visual axis is left open while confining the repair strictly to the medial canthal and nasal areas.

SUMMARY

We used a free tarsal-conjunctival graft from the upper eyelid and a nasal-based pedicle skin-muscle flap for reconstruction of the medial aspect of the lower eyelid. This had the advantage of confining the reconstruction to the local area and eliminated the need for a temporal flap or an eyelid sharing procedure that necessitates occluding the fissure for several weeks.

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FROZEN SECTION CONTROL IN THE SURGERY OF BASAL CELL CARCINOMA OF THE EYELID

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The recurrence rate after surgical excision of basal cell carcinoma of the eyelid has been variously reported as 0, 10, and 20%.[1-4] Einaugler and Henkind,[5] and Rakofski[2] reported that 50% of the excised tumors they studied had involvement at the margins. Aurora and Blodi[6] found, in all types of basal cell carcinoma, 23% incomplete excisions but only 16% among the solid-type lesions.

It may be that the difference in the reported recurrence rates reflects different methods of surgery and, therefore, the completeness of excision. We report herein the reliability of preoperative and intraoperative clinical evaluation of the amount of tissue to resect in basal cell carcinoma of the eyelid, and the recurrence rate after complete excision as determined by intraoperative frozen-section examination.

MATERIAL AND METHODS

We examined the medical records of 53 consecutive private patients with basal cell carcinoma of the eyelid. We concentrated on the size and site of the lesion, its duration, and the age of the patient when first seen.

Most of the patients (37; 69.8%) had a biopsy of the lesion preoperatively, which was studied with permanent sections. This was performed with the patient under local infiltrative anesthesia using 2% lidocaine (Xylocaine) with epinephrine. A quadrilateral pyramid approximately 2 to 3 mm on all sides was excised in depth with the skin surface as its base and with its apex deep in the lesion. Care was taken to avoid areas of the lesion that were obviously necrotic. After removal of the section, hemostasis was obtained either by using pressure, topical epinephrine, or light application of electrocautery. Of the 43 patients, six patients who had a small but cosmetically disfiguring lesion did not have a biopsy preoperatively. In these patients it was believed that the removal of the lesion with 3- to 4-mm clear margins (cancer surgery) would not be significantly more extensive than removal for purely cosmetic reasons; three patients who had recently been treated for basal cell carcinoma and who had no identifiable lesion at the treated site did not have a preoperative biopsy. These nine patients had either a full-thickness section of eyelid removed, including the lesion and 4 mm of normal-appearing tissue on all sides when an eyelid was involved, or a section of skin and subcutaneous tissue including the lesion and 4 mm of normal-appearing skin when the medial or lateral canthus was involved. The entire specimen was submitted and a frozen section was made through the lesion for diagnosis. If the lesion was a carcinoma, frozen sections were made of all the margins. If the lesion was benign, the defect was closed directly.

Patients who had a proven basal cell carcinoma by previous biopsy had a complete excision of the lesion as well as what appeared to be 3 to 4 mm of uninvolved (normal) tissue on all sides of the lesion.

If the tumor involved the eyelid, but

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not the canthi, a full-thickness section of eyelid was excised (Fig. 1, top left). If the tumor involved lateral or medial canthi with adjacent eyelid involvement, the specimen included full-thickness upper and lower eyelid, orbital tissue between the eye and orbital wall, and tissue over the orbital rim and outer orbital wall up to or including the periosteum (Fig. 2, top left). The depth of the tissue removed was determined by palpation of the tumor, and an attempt was made to remove at least a 3- to 4-mm section of tissue posterior to it.

The pathologist then entered the operating room and was shown the specimen, which had been excised but now was placed in its anatomic position on the patient's face. A full scale drawing of important anatomic landmarks, such as eyebrow and eyelids, was made on a piece of sterile cardboard (usually from a glove wrapper) with a methylene blue marking pen. The specimen was then placed in its proper anatomic position on the drawing and given directly to the pathologist. If there was any ambiguity about the specimen, the surgeon brought it to the laboratory and supervised the sectioning.

After excision, the pathologist studied all the margins of the specimen except for the eyelid margin, which was not a cut surface. When full-thickness eyelid had been removed, the pathologist cut a full-thickness (skin-to-conjunctiva) 3- to 4-mm

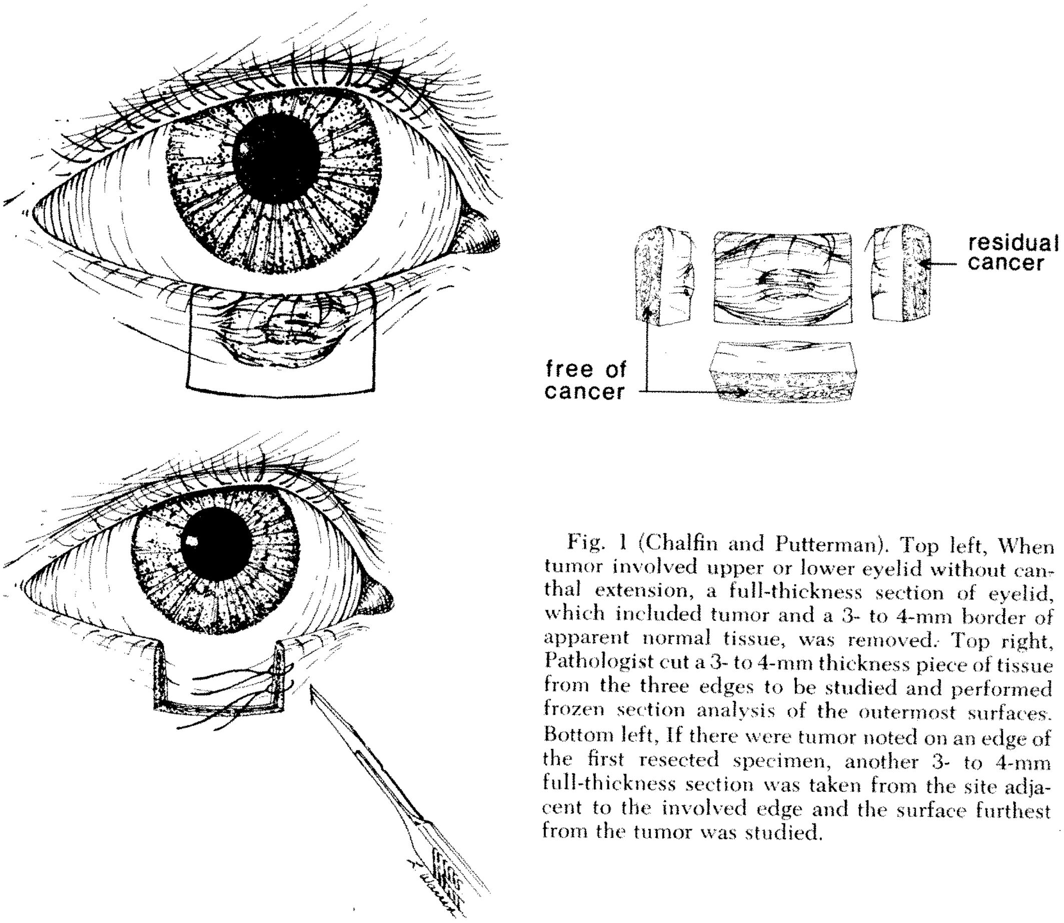

Fig. 1 (Chalfin and Putterman). Top left, When tumor involved upper or lower eyelid without canthal extension, a full-thickness section of eyelid, which included tumor and a 3- to 4-mm border of apparent normal tissue, was removed. Top right, Pathologist cut a 3- to 4-mm thickness piece of tissue from the three edges to be studied and performed frozen section analysis of the outermost surfaces. Bottom left, If there were tumor noted on an edge of the first resected specimen, another 3- to 4-mm full-thickness section was taken from the site adjacent to the involved edge and the surface furthest from the tumor was studied.

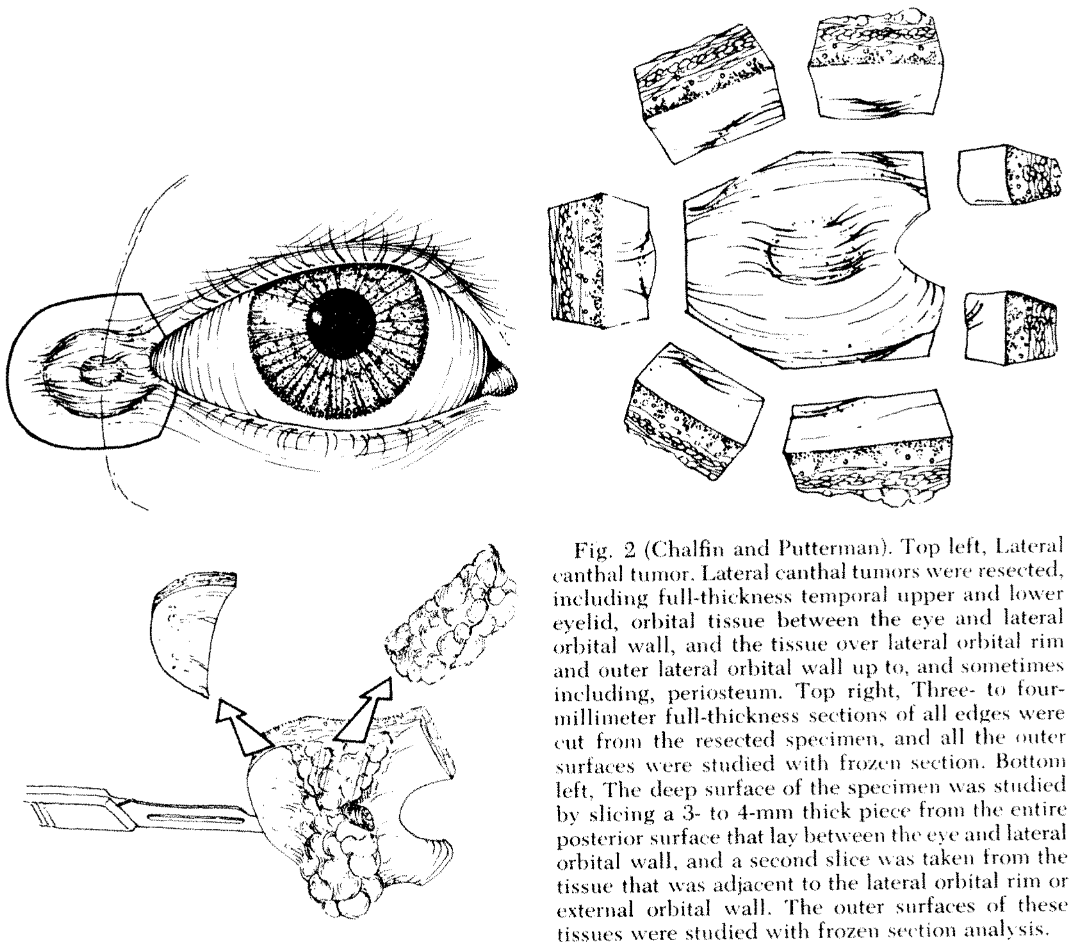

Fig. 2 (Chalfin and Putterman). Top left, Lateral canthal tumor. Lateral canthal tumors were resected, including full-thickness temporal upper and lower eyelid, orbital tissue between the eye and lateral orbital wall, and the tissue over lateral orbital rim and outer lateral orbital wall up to, and sometimes including, periosteum. Top right, Three- to four-millimeter full-thickness sections of all edges were cut from the resected specimen, and all the outer surfaces were studied with frozen section. Bottom left, The deep surface of the specimen was studied by slicing a 3- to 4-mm thick piece from the entire posterior surface that lay between the eye and lateral orbital wall, and a second slice was taken from the tissue that was adjacent to the lateral orbital rim or external orbital wall. The outer surfaces of these tissues were studied with frozen section analysis.

section from each of the three margins to be studied (Fig. 1, top). These tissues were then frozen and a thin sheet of tissue from the outermost side of the specimen was sliced and prepared for microscopic examination (Fig. 1, top right).

The lesion was then studied en bloc. Each entire margin was studied. The pathologist cut parallel to the margin and continued cutting sections until both the conjunctiva and skin edges were reviewed for the entire length of the margin being studied.

If there was any doubt of residual tumor in the studied edges, another surgical resection was performed over the suspected area (Fig. 1, top right and bottom). Two 6-0 black silk sutures were placed through skin and conjunctiva at the involved edge and then another 3- to 4-mm full-thickness section of eyelid was taken. Again, the surface furthest from the tumor and from the silk sutures was studied with frozen sections. The process was continued until all margins were free of tumor.

If the tumor involved the medial or lateral canthi, the upper and lower eyelid edges were studied as described for central eyelid tumors (Fig. 2, top). The entire thickness of the outer edges of the specimen were then cut in about 3 to 4 mm × 15 mm blocks of tissue, and all outer surfaces were studied (Fig. 2, top right).

The undersurface of the specimen was studied by slicing one 3- to 4-mm thick section from the surface of the tissue that existed between the eye and orbital wall and a second section from the tissue that had been adjacent to the orbital bone (Fig. 2, bottom). These sections were then frozen and the deeper or outer surfaces sliced and studied.

Pathologic reports were given not only on the presence of deep involvement, but also in what specific area the involvement existed. This often allowed the surgeon to spare structures (for example lacrimal sac) that might otherwise have to be excised.

Closure was attained by direct apposition of tissues whenever possible. Indirect methods included lateral canthotomy with or without cantholysis, semicircular skin incisions at the lateral canthus,[7,8] Cutler-Beard flaps,[9] skin grafts, or rotated skin flaps.

RESULTS

Patients with no previous history of basal cell carcinoma—Of the 37 patients on whom we performed the initial surgery, the mean age was just under 60.5 years and 51.4% (19) were female, (Table 1). In these patients, the right eyelid was involved more frequently than the left (19 vs 18, 51.4% vs 48.6%).

The distribution according to the site of the lesion was similar to that of other studies. Most lesions (23; 62.2%) involved the lower eyelid, followed by the medial canthus (7; 18.9%), the upper eyelid (6; 16.2%), and the lateral canthus (1; 2.7%).

The mean time before patients sought medical attention, when recorded, was just under 22 months; the range was from two to 120 months. More than half (20; 54.0%) of the patients sought help within 12 months.

Of the 37 patients in this group, 29 had preoperative biopsies. The eight who did not had small lesions that they wanted removed whether malignant or not. These lesions were removed with a surrounding border of normal tissue and biopsy was performed under frozen section. Because all of the patients described in this group had proved basal cell carcinoma, Table 1 in no way reflects the percentage of biopsies that are positive for malignancy.

All the lesions were excised with a border of at least 3 to 4 mm of normal or uninvolved tissue, as judged by the surgeon based on his preoperative and intraoperative examinations. Despite this evaluation, 15 (40.54%) of the patients had one margin that contained tumor cells, four (10.8%) had two positive margins, and one (2.7%) had three positive margins. Thus, a total of 20 (54.04%) of the patients studied had at least one involved margin when gross visual assessment of the tumor was used to judge the amount of tissue to be resected.

Patients with previously treated basal cell carcinoma—The mean age of the 16 patients who had basal cell carcinoma at sites previously treated by other physicians was 65.9 years and eight of them were male (Table 2). In these patients the left eyelid was involved more frequently than the right eyelid (11 vs 5, 68.8% vs 31.2%). Most of the lesions were in the lower eyelid (10; 62.5%), followed by the upper eyelid (4; 25%), and the medial canthus (2; 12.5%).

The mean time that the patients had the lesions was 27.9 months. This does not reflect how long the patient waited before seeking medical advice, because all these patients were treated during this interval. No effort was made to determine the time interval between previous treatment and reappearance of tumor because it was not certain whether these represented residual tumors or true recurrences.

Biopsies were performed on 11 of the 18 tumors listed in Table 2. Patient Nos. 38 and 39 had two separate tumors, which account for 18 tumors in 16 patients. The seven who did not have a preoperative

TABLE 1

PATIENTS WITH NO PREVIOUS HISTORY OF BASAL CELL CARCINOMA OF THE EYELID*

Patient No., Age (yrs), Sex	Site	Duration (mos)	Biopsy	Number and Site of Positive Frozen Sections	Follow-up (mos)	Evidence of Recurrence
1,60,M	RUL	12	Yes	1 (temporal)	18	—
2,65,M	LMC	6	Yes	0	14	—
3,67,F	RLL	24	No	1 (nasal)	6	—
4,81,F	LMC	18	Yes	1 (temporal)	12	—
5,58,F	LUL	24	Yes	2 (nasal, temporal)	12	—
6,55,F	LLL	2	Yes	0	20	—
7,46,M	RLL	?	Yes	2 (inferior, deep)	50	—
8,43,M	RLL	?	No	2 (superior, inferior)	5	—
9,58,M	LLL	?	Yes	1 (deep)	10	—
10,54,F	LLL	2	Yes	1 (nasal)	18	—
11,65,M	RLL	18	Yes	1 (nasal)	3	—
12,52,M	RLL	4	Yes	0	5	—
13,77,F	RLL	6	Yes	0	10	—
14,75,F	RL	60	Yes	0	6	—
15,53,M	RLL	12	Yes	1 (nasal)	9	—
16,50,F	RLL	3	Yes	3 (nasal ×2, temporal)	32	—
17,86,M	LLL	7	Yes	2 (nasal, deep)	6.5	—
18,75,F	RUL	24	No	1 (temporal)	18	—
19,55,F	LUL	40	Yes	0	10	—
20,81,F	LLL	5	No	0	6	—
21,?,F	LMC	24	No	1 (temporal)	32	—
22,54,F	RLL	6	Yes	0	23	—
23,31,F	RLL	12	No	0	48	—
24,76,M	RMC	6	Yes	1 (nasal)	6	—
25,33,M	LUL	24	Yes	0	18	—
26,66,F	RLL	?	No	0	2	—
27,68,F	LLL	Many years	Yes	0	6	—
28,50,F	RLL	120	Yes	0	3	—
29,73,F	RLL	120	Yes	1 (temporal)	6	—
30,34,M	LMC	3	No	0	†	†
31,73,M	RLL	2	Yes	1 (nasal)	†	†
32,58,F	LLL	24	Yes	1 (nasal)	†	†
33,54,M	LLL	?	Yes	0	†	†
34,64,F	RMC	36	Yes	1 (temporal)	†	†
35,76,M	LMC	1.5	Yes	1 (temporal)	†	†
36,54,M	LUL	8	Yes	0	†	†
37,57,M	LLL	?	No	1 (inferior)	†	†
Mean,60,47		21.8			14.3	

*RUL designates right upper eyelid; RMC , right medial canthus; RLL, right lower eyelid; RLC, right lateral canthus; LUL, left upper eyelid; and LMC, left medial canthus; ?, unknown.

†These patients had their surgery within two months of this report. Follow-up data and the presence of recurrences are unavailable at this time.

TABLE 2

PATIENTS WITH PREVIOUS TREATMENT OF BASAL CELL CARCINOMA WITH EVIDENCE OF A RECURRENCE*

Patient No., Age (yrs), Sex	Site	Duration (mos)	Biopsy	Number and Site of Positive Frozen Sections	Follow-up (mos)	Evidence of Recurrence
38,73,F	LLL	48	Yes	0	39	—
39,84,F	LUL	24	Yes	0	21	—
40,86,M	LLL	?	Yes	0	13	—
41,51,F	RLL	18	Yes	1 (conjunctiva)	36	—
42,78,M	LLL	48	Yes	0	44	—
43,56,M	RLL	7	No	0	13	—
44,78,M	LLL	90	No	3 (nasal, inferior superior)	30	—
45,77,M	LLL	24	No	0	12	—
46,52,F	RLL	3	Yes	1 (nasal)	2	—
47,66,F	RUL	?	Yes	6	52	—
48,81,M	LLL	10	Yes	1 (temporal)	5	—
49,63,F	LUL	3	No	1 (nasal)	41	—
50,84,M	RLL	4	No	1 (nasal)	3	—
51,64,M	LMC	?	Yes	1 (temporal)	5	—
52,62,F	LMC	24	No	2	†	†
53,60,F	LUL	60	No	0	†	†
Mean,65,9		27.9			22.6	

*RUL designates right upper eyelid; RMC, right medial canthus, RLL, right lower eyelid; RLC, right lateral canthus; LUL, left upper eyelid; and LMC, left medial canthus; ?, unknown.

†These patients had their surgery within two months of this report. Follow-up data and the presence of recurrences are unavailable at this time.

biopsy had no identifiable lesions in recently treated sites.

Six of the excised tumors (33.3%) had one positive margin; one patient (5.5%) had two positive margins, and another patient (5.5%) had three positive margins. Patient No. 47 had an extensive tumor that involved most of the left side of her face. Repeated specimens were taken on all sides of the lesion until all the tumor except for the inferior margin of the excision, which now extended far into her cheek, had been removed (Fig. 3). This area was later treated by Mohs' chemosurgery.[10] Thus, nine of 18 (50%) tumors were incompletely excised when gross visual assessment of the extent of the tumor was used to judge the amount of tissue to be resected.

Both patient groups were asked to return for examination for recurrence of tumor every three months for a year, followed by every six months for another two years, and yearly thereafter. In most cases, the patients were discharged to the care of the referring ophthalmologist after postoperative healing was complete. The referring ophthalmologist then assumed responsibility for the follow-up care of the patient and the same reexamination sequence was advised. After three months postoperatively, 18 of the 43 patients (41.9%) were subsequently followed up by us and 25 (58.1%) were followed up by the referring ophthalmologists. A questionnaire was sent to each referring ophthalmologist to ask when they had seen the patient last, whether or not there was evidence of a recurrence, and the disposition of any recurrences noted. All questionnaires were answered and returned.

Follow-up among the 29 patients we

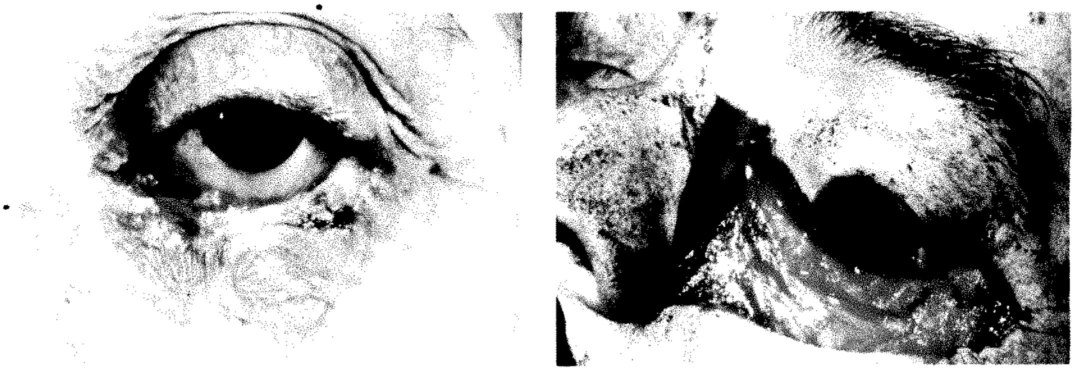

Fig. 3 (Chalfin and Putterman). Patient 47. Left, Left lower eyelid lesion. Right, Same patient after excision of all tumor, except nasal and inferior borders as shown by frozen section. Mohs' chemosurgery was used to treat these two borders.

treated for primary eyelid basal cell carcinoma more than three months before this report ranged from two to 50 months with a mean of 14.3 months. Seventeen (58.6%) were followed up for at least nine months after surgery. These values represent the interval between the date of surgery and the most recent examination of the patient. No patient whom we treated for a previously untreated basal cell carcinoma of the eyelid by complete excision of the tumor under frozen section control had recurrence of the tumor.

Follow up among the 14 patients whom we treated for a recurrence of basal cell carcinoma of the eyelid previously treated by another physician ranged from two to 52 months. The mean follow-up time was 22.6 months; ten (71.4%) were followed up for at least 12 months after surgery. There was no recurrence of the tumor after it had been excised completely under frozen section control.

DISCUSSION

The conflicting reports of the recurrence rate of basal cell carcinoma of the eyelid probably reflect differences in the technique of tumor excision and, therefore, the completeness of the excision. We believe that it is necessary to study all the margins of the excised tissues completely to ensure total excision of tumor cells.

In the 53 patients described in this paper, visual assessment of the tumor, using both the slit lamp and loupe on

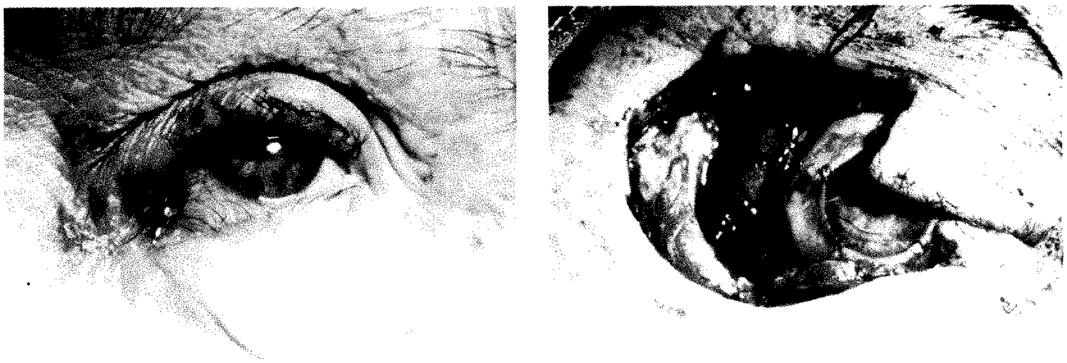

Fig. 4 (Chalfin and Putterman). Patient 4. Left, Left medial canthal tumor. Right, Same patient after excision of all tumor, as shown by frozen section.

many occasions, was reliable only about 50% of the time, even though the excision included at least 3 to 4 mm of normal-appearing skin at every margin. Often small-appearing lesions required a large resection to remove all tumor cells (Figs. 3 and 4).

None of the patients who had a complete excision of the tumor had a recurrence of the tumor. We believe that the lack of recurrence is because of the complete removal of tumor cells, and that this can only be accomplished with frozen section control of the excision.

SUMMARY

Two groups of patients, 37 with primary basal cell carcinoma of the eyelid and 16 with basal cell carcinoma of the eyelid previously treated by other physicians were reviewed. All of the tumors were excised with at least 3 to 4 mm of normal-appearing tissue at each margin of resection. All of the excised specimens were submitted for frozen section studies of the margins. A total of 20 (54.04%) of the previously untreated tumors and eight of the previously treated tumors had at least one margin involved with tumor cells. Repeated excisions under frozen section control were done until all of the margins were free of tumor cells. To date, none of the patients treated in this manner had a recurrence of the tumor.

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INVERTED FOLLICULAR KERATOSIS

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Inverted follicular keratosis is a benign skin lesion that occurs in middle-aged and older individuals, which mimics malignant lesions, especially squamous cell carcinoma, both clinically and pathologically.[1-10] Although this information is included in descriptions of inverted follicular keratosis in numerous textbooks, few reports have been published, nor has the incidence of inverted follicular keratosis in an ophthalmic series been determined. We report herein our experience with inverted follicular keratosis and compare its incidence with that of other eyelid lesions.

MATERIAL AND METHODS

All skin lesions defined as inverted follicular keratosis on file here during the period from 1966 to 1976 were reviewed. Additionally, all eyelid lesions received in the laboratory during 1975 were reviewed in order to determine the relative incidence of inverted follicular keratosis.

RESULTS

In 17 patients, 17 lesions of inverted follicular keratosis were identified (Fig. 1). The median age of the patients was 69 years (range, 46 to 93 years). The lesions were distributed randomly between upper and lower eyelids. The median duration of the lesions in this series before surgery was six months. Two of these lesions were present and stable for 20 and 50 years respectively, and showed significant growth just before surgery. Clinical diagnoses were verruca (four), papilloma (four), skin lesion (three), cutaneous horn (two), granuloma (one), senile keratosis (one), carcinoma (one), and malignant melanoma appearing as a cutaneous horn (one).[9] All of the lesions were characterized by a papillomatous configuration. Most lesions were small with a median size of 6 × 5 × 4 mm (range, 1 × 0.5 × 0.5 mm to 20 × 8 × 4 mm). Follow-up data were obtained on 14 of the 17 patients. The median period of follow-up was 19 months (range, one week to six years) and no lesions recurred.

Histologically, all specimens were characterized by acantholysis and squamous eddy formation (Fig. 2). In two

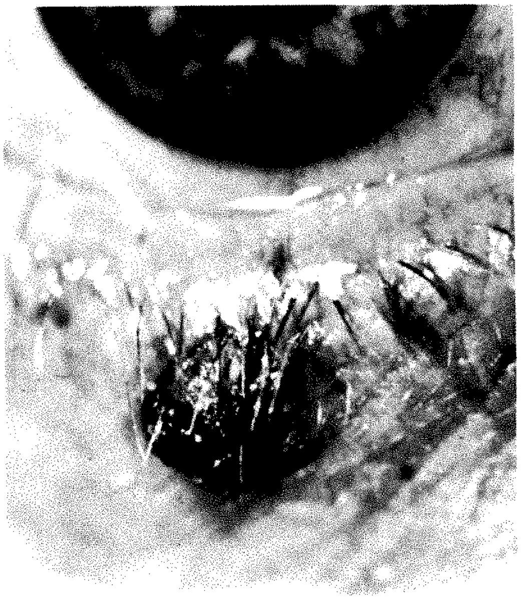
Fig. 1 (Sassani and Yanoff). A typical clinical appearance of inverted follicular keratosis.

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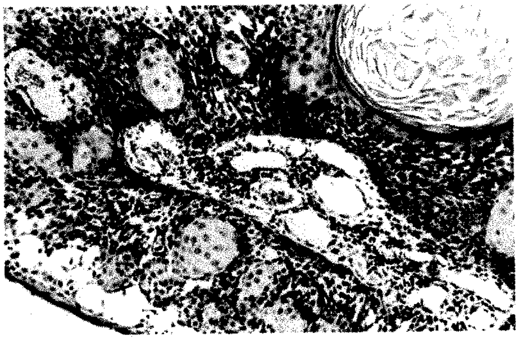

Fig. 2 (Sassani and Yanoff). Acantholysis (separation of epithelial cells) and squamous eddies (solid balls of epithelial cells) (hematoxylin and eosin, ×101).

specimens the acantholysis had progressed to intraepithelial cyst formation (Fig. 3). Additionally, all lesions showed acanthosis and hyperkeratosis (Fig. 4). All lesions showed a subepithelial inflammatory infiltrate of lymphocytes and plasma cells. The infiltrate was mild in seven cases, moderate in eight, and marked in two. Parakeratosis was noted in 13 lesions and mitotic figures in ten. Three of the lesions were pigmented because of dendritic melanocytes.

The Table lists the histologic diagnoses of 149 eyelid specimens received here in 1975. Three (2%) of the lesions were inverted follicular keratosis.

Discussion

Helwig[5] described a benign lesion that sometimes clinically and histologically mimicked carcinomas, especially squamous cell carcinoma. Since then, there have been several reports that further delineated the characteristics of inverted follicular keratosis.[1-4,6-10] Duperrat and Mascaro[4] were the first to suggest an origin specifically from the infundibulum of the hair follicle, a concept supported by others.[1,7]

In general, inverted follicular keratosis is found in individuals middle-aged or older; however, the median age in our series was 69 (range, 46 to 93 years). Other investigators have reported median ages from 50 to 70 years. In our study the lesions were found four times more frequently in men than in women. Most lesions that we evaluated were small, which is typical of inverted follicular keratosis.

Inverted follicular keratosis is not typi-

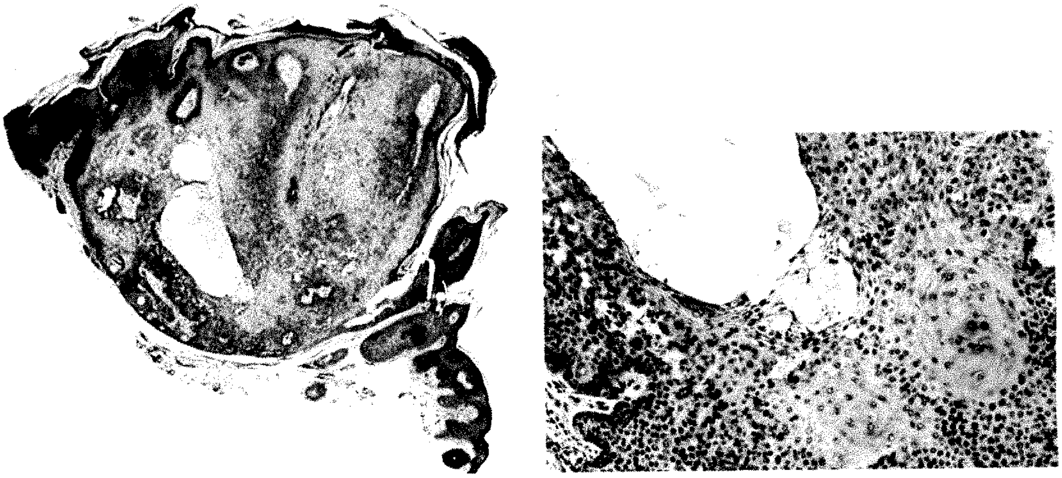

Fig. 3 (Sassani and Yanoff). Left, Large intraepithelial cyst within papillomatous lesion is a result of confluent acantholysis (hematoxylin and eosin, ×16). Right, Higher magnification shows cyst, acantholysis, and squamous eddy formation (hematoxylin and eosin, ×101).

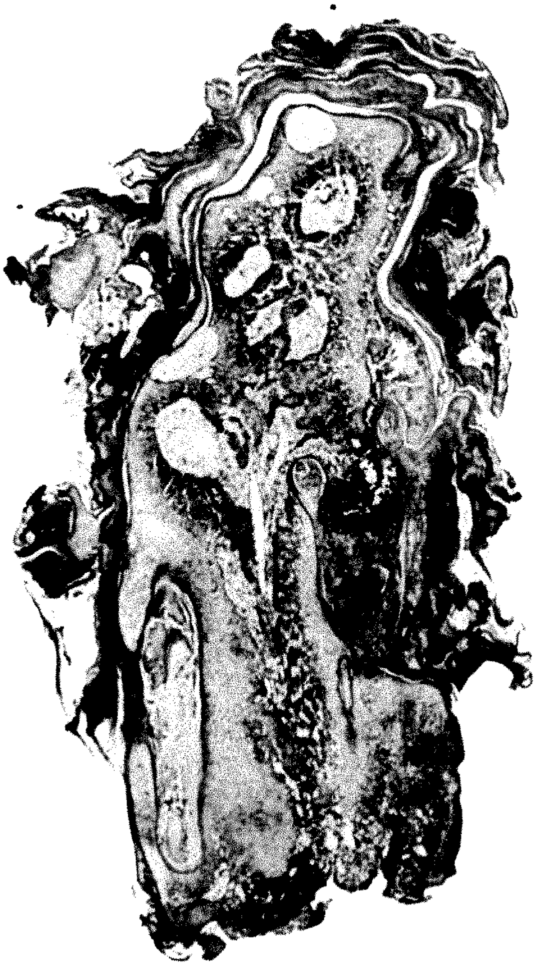

Fig. 4 (Sassani and Yanoff). Marked hyperkeratosis and acanthosis is present in papillomatous lesion. Clinical diagnosis had been cutaneous horn (hematoxylin and eosin, ×16).

TABLE

DIAGNOSIS OF 149 EYELID LESIONS RECEIVED IN LABORATORY OF OPHTHALMIC PATHOLOGY IN 1975

Diagnosis	No. of Cases
Chalazion	24
Basal cell carcinoma	21
Squamous papilloma	17
Seborrheic keratosis	15
Epithelial inclusion cyst	12
Other inflammatory lesions	10
Xanthelasma	10
Fibroepithelial papilloma	9
Nevus	7
Other benign noninflammatory lesions	6
Actinic keratosis	5
Verucca vulgaris	4
Inverted follicular keratosis	3
Other benign cysts	3
Keratoacanthoma	2
Sebaceous gland carcinoma	1

cally an eyelid lesion. One study showed that 34 of 40 inverted follicular keratosis lesions appeared predominately on the face, but only two appeared on the eyelids or eyebrow.[7] No patient in our series had a recurrence of inverted follicular keratosis; it has been known to recur rarely.[3] Multiple lesions also may be found.[3]

Pathologically, our lesions were similar to those reported by others in that they were typified by acantholysis and squamous eddy formation. Most investigators have pointed out that inverted follicular keratosis rarely is pigmented, but three of the 17 lesions in our study contained pigment. Mitotic figures were found in ten of the 17 lesions, but none was atypical and no other histologic findings of malignancy were noted. Additionally, information obtained at follow up showed the benign nature of inverted follicular keratosis.

SUMMARY

We reviewed 17 cases of inverted follicular keratosis. The median age of the patients at the time of surgery was 69 years. Follow-up in 14 cases showed no recurrences of inverted follicular keratosis, which is a benign skin lesion, often mistaken clinically and pathologically for a malignancy. Inverted follicular keratosis is characterized histologically by the presence of squamous eddies, acantholysis, acanthosis, and hyperkeratosis.

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INTRAOCULAR PENETRATION OF TRIFLURIDINE

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One of the greatest problems encountered when using antimetabolite drugs in viral infections is loss of therapeutic effect because of poor tissue penetration and rapid conversion to less active or totally inactive metabolites. Topical trifluridine appeared to be effective in treatment of clinical herpetic iritis, suggesting intraocular penetration of this drug in therapeutic levels.[1,2] More recently [3] in vitro studies using isolated rabbit corneas mounted in a lucite block perfusion system have shown that both trifluridine and idoxuridine penetrate through the cornea from the epithelial side at a greater rate than vidarabine (Ara-A), and that this rate may be increased by two-fold for trifluridine and idoxuridine and five-fold for vidarabine if the epithelium is absent. The sole breakdown products identified on the aqueous humor side of the chamber were 5-carboxy 2′-deoxyuridine for trifluridine, 2′-deoxyuridine and iodouracil for idoxuridine, and hypoxanthine arabinoside for vidarabine. The antiviral activity of 5-carboxy 2′-deoxyuridine is less than that of trifluridine (Burroughs Wellcome Co., personal communication, 1978); hypoxanthine arabinoside is approximately 20% as active as vidarabine,[4] and 2′-deoxyuridine and iodouracil are biologically inactive.

Trifluridine is considerably more water soluble (5.0 mg/100 ml) than either vidarabine (0.5 mg/100 ml) or idoxuridine (0.1 mg/100 ml), and it has greater potential for achieving therapeutic levels of unmetabolized drug in the aqueous humor of the in vivo system.

Clinical studies have indicated that topical trifluridine might be an effective treatment for herpetic iritis but in vitro studies have indicated that trifluridine is not found on the endothelial side of the rabbit cornea.[3] We report herein a study of aqueous humor samples taken from patients at the time of surgery to compare levels of antimetabolites in those patients receiving trifluridine with those who received only the usual preoperative antibiotic medication.

MATERIAL AND METHODS

Five normal patients undergoing cataract extraction received gentamicin drops every 30 minutes × 4 up to 30 minutes preoperatively. Five patients with a history of ocular herpes and corneal scarring undergoing corneal transplantation received gentamicin as did the normal cataract patients, but they also received 1% trifluridine ophthalmic solution (Viroptic), 1 drop every 10 minutes × 4 in the operating room, the last drop being given five minutes before sterile preparation of the eye. In all patients the forniceal cul-de-sacs were swept dry with Weck cell

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From the Department of Cornea Research, Eye Research Institute of Retina Foundation, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts (Dr. Pavan-Langston), and Wellcome Research Laboratories, Research Triangle Park, North Carolina (Dr. Nelson). This study was supported in part by Burroughs Wellcome Company; Abbe N. Prince Fund; and in part by the Massachusetts Lions Eye Research Fund, Inc.

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sponges immediately before aqueous aspiration. The syringe was sealed and immediately frozen at −70C until time for analysis by high pressure liquid chromatography on a reversed phase-ODS column with two ultraviolet absorption detectors connected in series set at 254 nm and 280 nm, respectively. Minimum detectable levels of trifluridine and 5-carboxy 2′-deoxyuridine were approximately 2 μM or approximately 0.5 μg/ml.

RESULTS

The intraocular concentrations of trifluridine and the metabolite, 5-carboxy 2′-deoxyuridine, in comparison to untreated controls are shown in the Table. The chromatograms of the controls had no interfering peaks in the position corresponding to the compounds of interest that made the analysis uncomplicated. In the five patients receiving trifluridine preoperatively, none had detectable levels of 5-carboxy 2′-deoxyuridine, whereas four out of five patients had a measurable level of intact trifluridine. Case 9 who did not have a detectable level of trifluridine, had relatively healthy, intact epithelium with only minimal roughening of the surface and a scarred but normal stromal thickness (Fig. 1). In Case 10, a level of 3.1 μM trifluridine was found despite the presence of excellent epithelium. This concentration of trifluridine was probably caused by extreme corneal stromal thinning (about one-eighth normal thickness) (Fig. 2). This patient had had a corneal perforation 18 months previously that was treated with cyanoacrylate glue. Subsequent corneal healing had dislodged the adhesive spontaneously eight months preoperatively. Case 8 had a moderate

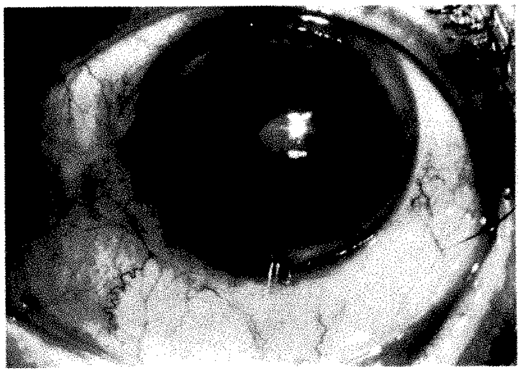

Fig. 1 (Pavan-Langston and Nelson). Case 9. Slightly roughened but intact corneal epithelium with scarred but normal thickness stroma.

TABLE

INTRAOCULAR PENETRATION OF 1% OPHTHALMIC SOLUTION OF TRIFLURIDINE INTO THE HUMAN AQUEOUS HUMOR

Case	Epithelium Condition	Stroma Condition	Iritis	Trifluridine* (μM)	5-carboxy 2′-deoxyuridine* (μM)
Cataracts, Cases 1-5†	Good	Normal	0	< 2	<2
HSV-PK‡-6	Fair	Edema	0	13.6	<2
HSV-PK‡-7	Poor	Faceted	0	43.9	<2
HSV-PK‡-8	Fair	Glued descemetocele	+	6.4	<2
HSV-PK‡-9	Good	Moderate scar	0	< 2	<2
HSV-PK‡-10	Good	Very thin	0	3.1	<2

*The concentrations of trifluridine and 5-carboxy 2′-deoxyuridine in the aqueous humor were determined by high pressure liquid chromatography.

†Cataract patients, Cases 1-5, received no trifluridine.

‡HSV-PK designates herpes simplex virus-penetrating keratoplasty; patients received trifluridine, 1% ophthalmic drops, immediately before surgery.

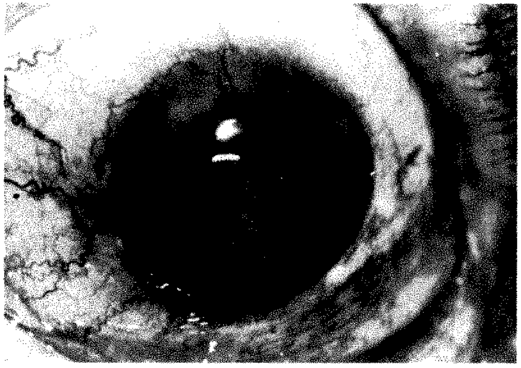

Fig. 2 (Pavan-Langston and Nelson). Case 10. Healthy intact epithelium over markedly thinned, scarred stroma.

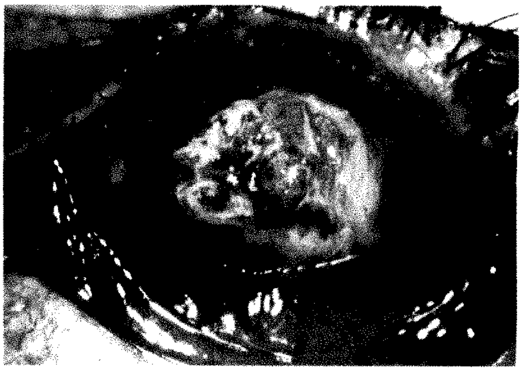

Fig. 3 (Pavan-Langston and Nelson). Case 8. Cyanoacrylate adhesive covering descemetocele in eye with active iritis.

were submitted for frozen section studies of the margins. A total of 54.04% (20) of the corneal surface had not been covered with cyanoacrylate adhesive to prevent corneal perforation of a descemetocele. This was the only actively inflamed eye at the time of surgery (Fig. 3). Case 6 had an aqueous humor concentration of trifluridine of 13.6 μM. This patient had unhealthy but intact gray edematous epithelium over a failed edematous (twice normal stromal thickness) penetrating keratoplasty (Fig. 4). Case 7 displayed the highest level of trifluridine, 43.9 μM, probably because of the unhealthy disrupted epithelium over a somewhat thinned and faceted stroma (Fig. 5).

DISCUSSION

In 1972 Pavan-Langston and associates[5] reported the apparent amelioration of herpetic iritis after topical vidarabine therapy. The resulting clinical study on the intraocular penetration of vidarabine and idoxuridine indicated that trace amounts of the parent compound, vidarabine, and its metabolite, hypoxanthine arabinoside, were present in human aqueous humor 20 minutes after application of 3% ointment if the epithelium were unhealthy or ulcerated. Patients receiving idoxuridine had only a uracil derivative of the antimetabolite and no unmetabolized drug regardless of the epithelial status.

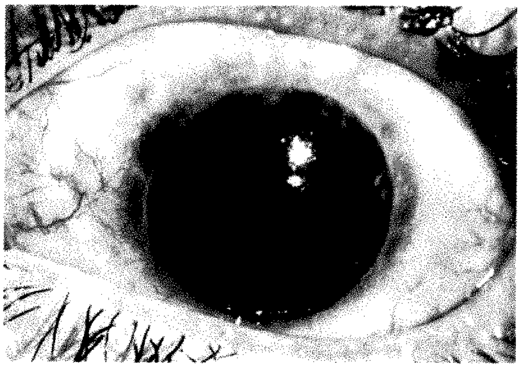

Fig. 4 (Pavan-Langston and Nelson). Case 6. Gray, edematous, intact epithelium over swollen, failed herpetic graft.

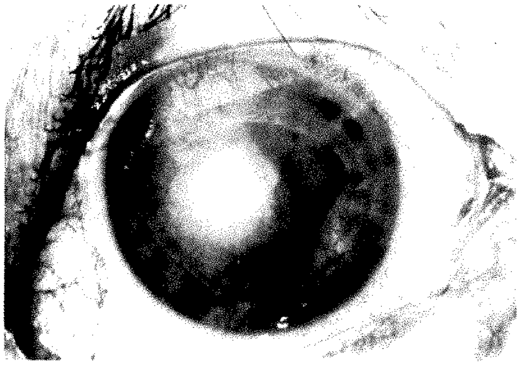

Fig. 5 (Pavan-Langston and Nelson). Case 7. Edematous, disrupted epithelium over thinned, scarred stroma.

In the present study, levels of intact trifluridine ranging between 3.1 to 43.9 μM were found in patients with unhealthy epithelium or marked stromal thinning. The metabolic by-product of trifluridine, 5-carboxy 2′-deoxyuridine, was not found in any patient's aqueous humor. Unlike in vitro studies by O'Brien and Edelhauser,[3] no metabolic breakdown products of any form were detected in humans.

Studies by Collins and Bauer[6,7] comparing the activity of herpes virus inhibitors revealed that the 50% effective dose for trifluridine was 0.75 to 1.81 μM by the plaque reduction method for type 1 and type 2 herpes simplex virus, and 15.13 to 44.66 μM by the yield reduction method for type 1 and type 2 herpes simplex virus. No absolute differences were observed in type sensitivity, although type 2 strains were generally less sensitive than were type 1 strains. The aqueous humor levels of intact drug in the present study are within these concentration ranges, particularly when compared with the results from the plaque reduction method, in any patient with minimal disruption of the corneal epithelium or stromal thinning, or both.

Drugs that penetrate the eye well must have good biphasic solubilities.[8] Water solubility is necessary for tear film transport and stromal penetration, whereas lipid solubility is necessary to cross cell membranes. Vidarabine and idoxuridine have relatively poor biphasic solubility although the higher water solubility of vidarabine's metabolite, hypoxanthine arabinoside, is probably responsible for this drug reaching the aqueous humor in detectable concentrations. The phosphorylated derivative of vidarabine, adenine arabinoside monophosphate, also has high water solubility. Falcon and Jones[8] reported that this drug is extremely effective when given as a single drop a day in treatment of experimental herpetic epithelial keratitis because it enters damaged, but not healthy, epithelial cells effectively. This is supported by Carrasco's[9] report of virus-induced changes in host cell plasma membranes that make normally impermeable cells readily permeable to poorly soluble antimetabolites. Adenine arabinoside monophosphate's water solubility is probably the factor responsible for the severe stromal toxicity, as well as the iritis noted in wound healing studies when the drug was used eight times a day for one to three weeks.[10]

Trifluridine has good solubility in both water and lipids that probably enhances rapid transport of the intact drug across the entire cornea.[7] This superior biphasic solubility accounts for the levels of trifluridine in aqueous humor samples examined in the present clinical study. Unlike idoxuridine and vidarabine, it is possible with 1% trifluridine topical drugs to achieve therapeutically significant levels of the active antiviral drug at an intraocular site where intact virus may replicate in tissues beyond the reach of other antivirals. This represents a therapeutic advance that would be advantageous to clinicians faced with the problematic diseases of herpes simplex stromal interstitial keratitis and iritis.

SUMMARY

We studied intraocular penetration of topically applied trifluridine in five patients with herpetic keratitis undergoing penetrating keratoplasty. We compared the concentration of trifluridine and its metabolite 5-carboxy 2′-deoxyuridine in the aqueous humor to those of normal control patients undergoing routine cataract extraction without preoperative antiviral therapy. Significant concentrations of intact trifluridine were achieved in the aqueous humor after topical application of 1% trifluridine ophthalmic drops. The metabolite, 5-carboxy 2′-deoxyuridine, was not found in the aqueous humor.

Unlike idoxuridine and vidarabine, it is possible to achieve therapeutic levels of trifluridine at intraocular sites that would be advantageous in the treatment of deep herpetic disease involving the stroma and iris.

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CONTRAINDICATION TO THE USE OF OCULAR PHENYLEPHRINE IN IDIOPATHIC ORTHOSTATIC HYPOTENSION

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Phenylephrine ophthalmic solution is frequently used to achieve adequate mydriasis.[1] It has long been used with a minimum of side effects, but a recent review from the National Registry of Drug-Induced Ocular Side Effects has raised the possibility that adverse reactions may be more common and more serious than was previously recognized.[2] Subgroups that seemed particularly susceptible to increased risk of the drug included those with cardiac disease, hypertension, aneurysms, advanced arteriosclerosis, and patients who are young or old.

Idiopathic orthostatic hypotension is an uncommon but dramatic syndrome in which there is dysfunction of the sympathetic nervous system and reduced plasma and urinary cathecholamines. This condition should lead to denervation hypersensitivity. We report herein the effect of the ocular administration of phenylephrine in subjects with this syndrome and in normal subjects.

SUBJECTS AND METHODS

Four patients with severe idiopathic orthostatic hypotension and eight normal subjects were studied. The patients ranged in age from 58 to 72 years; the normal subjects from 26 to 70 years. All of the patients and seven of the eight normal subjects were male. The normal subjects had no evidence of cardiac or vascular disease, and were not taking medications. The patients had not been taking any medications for at least three weeks before the study.

Diagnosis was confirmed by standing blood pressures of 60/0 mm Hg or less, lack of evidence of central nervous system dysfunction, and subnormal circulating and urinary catecholamines. Three of the four patients had increased supine blood pressures. Denervation hypersensitivity was confirmed by stepwise increasing intravenous boluses of phenylephrine until an increase in pressure of 25 mm Hg was achieved. This pressure increment was achieved in the idiopathic orthostatic hypotension patients with less than 20% of the dose required in the normal subjects.

Because hypersensitivity was suspected, the 2.5% (rather than 10%) phenylephrine HCl solution (NeoSyneprhine 2.5%), was used in a topical ocular application. This is a sterile, isotonic, buffered, low surface tension vehicle with sodium phosphate, sodium biphosphate, boric acid, and benzalkonium chloride, USP, 1:7,500. With the subjects in a comfortable reclining position, blood pressure was monitored with a sphygmomanometer at three minute intervals in the right arm. Blood pressure and heart rate were monitored for 30 minutes before and two hours after phenylephrine administration.

After administration of proparacaine HCl (Ophthaine), one drop in each eye for topical anesthesia, one drop of the 2.5% phenylephrine solution was admin-

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istered on the upper corneoscleral limbus in each eye. Intraocular pressure in each eye was monitored before and at 30-minute intervals after phenylephrine by Schiøtz tonometry. Proparacaine anesthesia was repeated at one-hour intervals.

RESULTS

No significant effects on blood pressure were seen in the normal subjects, and although there was a trend for heart rate to be reduced slightly, this did not reach statistical significance (Fig. 1). In marked contrast, the patients with idiopathic orthostatic hypotension had a dramatic increase in blood pressure (Fig. 2). The maximal pressor response in the four patients averaged 44/27 mm Hg. These subjects tended to have low baseline heart rates, and no further drop in heart rate with the phenylephrine was evident. No significant change in intraocular pressure was noted in either group.

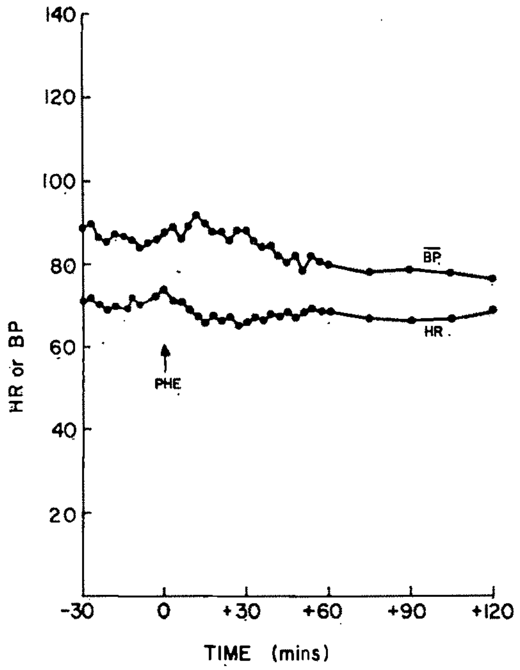

Fig. 1 (Robertson). Mean blood pressure and heart rate responses to 2.5% phenylephrine hydrochloride ophthalmic solution in eight normal subjects. One drop was placed in each eye.

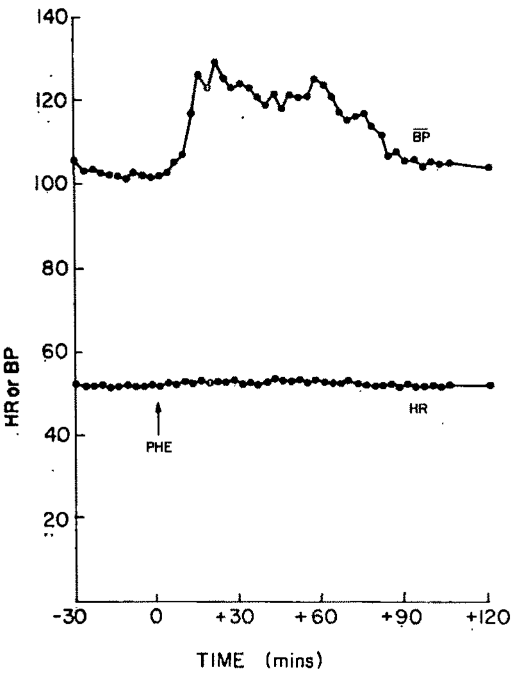

Fig. 2 (Robertson). Mean blood pressure and heart rate responses to 2.5% phenylephrine hydrochloride ophthalmic solution in four patients with idiopathic orthostatic hypotension. One drop was placed in each eye.

The increase in arterial blood pressure appeared within ten minutes and was present for at least 60 minutes after phenylephrine instillation. Subjective effects were mild, but two patients noted the prickly sensation that sometimes accompanies sympathomimetic therapy as the piloerector muscles are stimulated. One patient noted increased awareness of heartbeat. No abnormalities of rhythm were noted. Maximum blood pressures recorded during the hour after phenylephrine in the idiopathic orthostatic hypotension patients averaged 204/107 mm Hg. Changes were statistically significant in all four patients.

DISCUSSION

Phenylephrine HCl is an effective mydriatic.[1] As a potent alpha-adrenergic agonist, the 10% ophthalmic solution results in a 2-to-3-mm mydriasis without significant increase in intraocular pressure.[3]

The agent has been generally well tolerated, although occasional reports of pigment floaters have been published.[4]

In addition to such ophthalmologic side effects, cardiovascular complications have been sporadically encountered. When phenylephrine was systematically evaluated by Keys and Violante,[5] the threshold dosage was determined as 0.4 mg intravenously, 2.0 mg subcutaneously, and 50 mg orally. Because one drop of 10% ophthalmic solution contains 3.5 to 6.7 mg of phenylephrine, it would not seem likely that systemic effects of the drug would commonly occur.[6,7] However, in normal subjects, we and others[8] have observed pressor effects with smaller phenylephrine doses than those studied by Keys and Violante.

After their observation of a patient suffering a subarachnoid hemorrhage when a cotton pack soaked with 10% phenylephrine was applied to one eye, McReynolds, Havener, and Henderson[9] studied blood pressure effect of the topical application of 10% phenylephrine to hypertensive patients. Only six of these patients had a significant pressor response and it was mild, yet placement of six drops of this preparation into the conjunctival sac raises pressure 10 to 40 mm Hg.[10] Occasional reports of phenylephrine-induced hypertension have continued to appear,[7,10-12] particularly with respect to babies.[13,14] Seven additional cases have recently been reported by Fraunfelder from the National Registry of Drug-Induced Ocular Side Effects.[2] Other reactions, possibly related to phenylephrine included myocardial infarction, arrhythmias, and sudden death.[2] Patients who are young or old appeared to be most at risk of complication from the use of phenylephrine.

Idiopathic orthostatic hypotension is a degeneration of the autonomic nervous system that leads to a disabling postural hypotension. A syndrome of autonomic dysfunction associated with central nervous system abnormalities[15] and a syndrome of isolated autonomic insufficiency[16,17] have been described. Subjects with isolated autonomic insufficiency have reduced circulating and urinary catecholamines[17] and reduced stores of norepinephrine in blood vessels.[16] A hypersensitivity to exogenous norepinephrine has been observed.[16] A dramatic hypertensive response in these subjects to a 30-mg oral dose of phenylephrine has also been described.[18]

Our study documents extreme hypersensitivity to phenylephrine also when it is administered by the ocular route. The 44/27 mm Hg increase in blood pressure is especially striking because the usual dose of 10% phenylephrine used in practice would have delivered a fourfold greater amount of the drug. This would clearly have led to dangerously high blood pressure responses. Because idiopathic orthostatic hypotension usually occurs in the elderly population, the group in whom most untoward reactions have been seen, we believe that some of the adverse consequences of phenylephrine reported have occurred in patients with this disease.

The mechanism of this pressor response to ocular phenylephrine is undoubtedly partially caused by denervation hypersensitivity. However, the pharmacology of systemic absorption through mucosal surfaces is complex,[19] and the sympathetically denervated vessels of the patient with idiopathic orthostatic hypotension might allow greater drug absorption through mucosal surfaces than the sympathetically intact vessels of normal subjects. It is also possible that prior treatment with local anesthetic and disruption of the corneal epithelium by a Schiøtz tonometer could have resulted in increased drug absorption in patients with idiopathic orthostatic hypotension, but similar effects would be present in the normal group as well.

Whatever the mechanism of the increased pressor response to phenylephrine in idiopathic orthostatic hypotension, one should avoid giving routine doses of ocular sympathomimetics to patients with this condition.

SUMMARY

We studied the effect of a topical ocular application of 2.5% phenylephrine ophthalmic solution in patients with idiopathic orthostatic hypotension and in normal subjects. The normal subjects had no pressor effect to the drug. However, the patients with idiopathic orthostatic hypotension, had a marked pressor response. The maximal pressor response in the four patients averaged 44/27 mm Hg. The usual 10% phenylephrine used in practice could have delivered a fourfold greater amount of drug, and presumably would have led to dangerously high blood pressure responses.

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EFFECT OF BILATERAL AND UNILATERAL GRAFTS ON THE INCIDENCE OF REJECTIONS IN KERATOCONUS

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Keratoconus, an ectatic dystrophy of the cornea, is often managed by either spectacles or contact lenses. However, when keratoplasty must be used, the prognosis is favorable. Previous investigators reported an incidence of clear grafts ranging between 81 and 100%.[1-4] Chandler and Kaufman[5] reported an incidence of clear grafts in 91% of their cases with a 37% incidence of graft rejections. We report herein the results of 124 penetrating keratoplasties in 100 patients operated on for keratoconus (76 unilateral operations and 24 bilateral operations) at the Massachusetts Eye and Ear Infirmary between Jan. 1, 1971 and Dec. 13, 1975.

MATERIAL AND METHODS

We reviewed the records of 100 patients who underwent 124 penetrating keratoplasties at the Massachusetts Eye and Ear Infirmary between Jan. 1, 1971 and Dec. 31, 1975. The group consisted of 61 men and 39 women ranging in age from 11 to 69 years with an average age of 31 years. The follow-up period ranged from three months to five years with a mean of 18 months; only four patients had follow-up periods of less than one year. All preoperative eyes were white and quiet and the corneas were free from vascularization. The operating microscope was used in all procedures. The graft size varied between 7.5 and 9.0 mm with a mean of 8.5 mm. A continuous 10-0 monofilament nylon suture and rarely, 9-0 silk interrupted sutures were used to secure the donor button. There were 76 unilateral operations and 24 bilateral operations, and between the two groups, no significant differences with respect to age of donor tissue, preoperative handling of donor tissue, or surgical technique.

Graft rejections were diagnosed according to the following criteria[5,6]: (1) sudden onset of conjunctival injection in a previously uninflamed eye, (2) presence of graft edema, (3) presence of keratic precipitates on the endothelium, and (4) sudden onset of anterior chamber reaction. Rejections were confirmed independently by at least two different observers.

Graft rejections were treated with intensive topical corticosteroids consisting of 0.1% dexamethasone drops every hour for the first 24 to 48 hours and then tapered over the succeeding weeks as the rejection resolved.

RESULTS

In 76 eyes of patients grafted in only one eye for keratoconus there were ten rejections (13%) (Table 1). In the bilaterally grafted patients, there were 13 rejections in 48 eyes (27%). In both groups, rejection episodes were generally reversible, and only two grafts in each group eventually became opaque. The inci-

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TABLE 1

HOMOGRAFT REJECTIONS IN KERATOCONUS

Penetrating Keratoplasty	Total No. of Eyes	Homograft Rejections	Rejections* %
Unilateral transplants	76	10	13
Bilateral transplants	48	13	27

*P<.01

dence of rejection was not significantly influenced by differences in age, sex, graft size, type of suture used, or surgical complications in the unilateral or bilateral groups or between the group experiencing rejection and those free of rejection. The mean and median graft sizes were 8.5 mm in each group.

In the bilaterally transplanted cases, ten of 24 patients had at least one rejection episode and three patients had bilateral rejections. In nine cases, the second graft was rejected first, and in four cases, the first graft was rejected initially. No rejection occurred in the first graft before the performance of the second graft, at least one year after the first keratoplasty.

If the 24 eyes which were to receive eventual second grafts are included with the 76 unilateral eyes observed for one year after initial surgery, the rejection rate observed was ten of 100 (10%) (Table 2). Those 76 patients who did not receive second grafts showed no rejections in the second year; however, nine of the 24 patients (38%) who received additional transplants in the second eye demonstrated rejections. Thus, the incidence of immune reaction introduced by the second graft increased almost fourfold, and the risk for the first graft to develop an immune reaction increased from 0 to 17% in the year following the second graft (Table 2). Three out of the four rejections occurring in the first eye after a graft in the second were bilateral.

In the bilaterally grafted patients, the median rejection time was three months with a range of two weeks to 29 months, whereas in the unilaterally grafted patients, the rejection occurred slightly later at an average of 7.4 months with a median rejection time of eight months and a range of 1.5 to 12 months.

Continuance of corticosteroid treatment beyond the immediate postoperative period did not significantly decrease the incidence of homograft reactions in either group.

Vision was irreversibly lost during the postoperative period in only one case as a result of retinal detachment. In another case, vision was significantly impaired because of optic atrophy following uncontrolled secondary glaucoma. The only other serious complication in this series was the postoperative occurrence of posterior subcapsular cataracts.

Thirty-four (32%) of 107 eyes observed for at least eight months postoperatively and for which detailed records were available developed posterior subcapsular cataracts. There was no significant difference in the incidence of cataracts between the unilateral and bilateral groups.

The postkeratoplasty visual acuity improved significantly. Accurate final visual acuities were obtainable from records of 115 of the 124 keratoplasties studied. In 81 eyes with clear grafts and no cataracts, 70 (86%) achieved a visual acuity of 6/12 (20/40) or better. In the group of 34 patients who developed cataracts, 23 (68%) achieved 6/12 (20/40) visual acuity or better.

DISCUSSION

In our series, the incidence of graft rejection in 76 unilaterally grafted patients was 13%, but only two of the grafts were irreversibly lost. This is lower than the 37% incidence of graft rejection with 10% irreversible clouding previously reported by Chandler and Kaufman.[5] Additionally, the mean onset of rejection in this series occurred much later, eight months, as compared to eight weeks in their series.[5] Our incidence of 13% is more comparable to the incidence of 8% rejection reported by Keates and Falkenstein,[4] and 6% reported by Anseth.[7]

Graft reactions occurred twice as frequently in bilaterally grafted patients (27%) as in unilaterally grafted (13%), and the difference was statistically significant ($P<.01$) (Table 1). However, whereas only ten of the 100 patients grafted in one eye developed homograft reactions in the first year, nine of 24 (38%) of these patients receiving a graft in the second eye demonstrated subsequent rejection. Of those patients receiving second grafts, four rejected the first transplant, and three of these represented bilateral rejections. There was almost a fourfold increase in the risk of rejections in performing a second graft; the risk for the first graft to develop an immune reaction also increased from 0 to 17% (Table 2).

Keratoplasty in the second eye is not contraindicated, but because of these additional risks, we recommend that (1) a contact lens should be used in the second eye as long as possible; (2) two to three years should elapse before grafting the second eye; (3) excessively large second grafts should be avoided; and (4) corticosteroids should be used topically in both eyes during the immediate postoperative period.

Pouliquen and Rocher[8] also reported an increased incidence of homograft reactions in bilateral keratoplasty for a variety of diseases. However, early homograft reactions in both the unilateral and bilateral groups were easily reversed with intensive administration of topical corticosteroids. Only four homograft reactions (two in each group) were not reversed by corticosteroids; all four were subsequently regrafted without complication. The early diagnosis of homograft reaction is thus critical in determining the fate of the graft, as was emphasized by Chandler and Kaufman.[5]

TABLE 2

HOMOGRAFT REJECTIONS IN KERATOCONUS

Graft situation	1st postoperative year		2nd postoperative year	
	%	No.	%	No.
Unilateral	10	10/100	0	0/76
Bilateral				
Second graft	38	9/24	–	–
First graft	0	0/24	17	4/24

The major postoperative complication in our patients was the high incidence (32%) of posterior subcapsular cataracts. This presents a dilemma for the surgeon, for although homograft reactions pose a major threat to the clarity of the graft, surgical intervention for cataracts in young patients is hazardous with uncertain visual outcome and the potential disadvantage of monocular aphakia. Corticosteroids used postoperatively should be carefully monitored so as not to exceed a toxic dose for the lens. In our series, continuance of corticosteroid treatment beyond the minimum necessary to achieve a quiet eye did not decrease the incidence of homograft reactions in either group. Furthermore, if graft reaction did occur, it could easily be reversed when treated early. It is thus important during the postoperative period to perform careful slit-lamp examination of the lens to check for rejection. At the first sign of posterior subcapsular changes, topical corticosteroids should be eliminated or reduced to the lowest possible levels. Unfortunately, one may have to choose between furthering cataract formation and a continuous suppressive treatment of blocking homograft reaction to maintain graft clarity. Such decisions must always be made in relation to the visual needs and circumstances of the patient.

SUMMARY

We studied results of 124 keratoplasties in 100 keratoconus patients; 61 men and 39 women varying in ages from 11 to 69 years with an average age of 30 years. Twenty-four patients had bilateral grafts with a 27% incidence of rejection, and 76 patients had unilateral grafts with a 13% incidence of rejection ($P<.01$). Data analysis of the first year after the graft of first eyes and subsequent years after the graft of first and second eyes, however, revealed a nearly fourfold increase of rejections following second grafts, with a 17% increased chance of rejection for the first graft after the second was implanted. Only two of the bilateral and two of the unilateral grafts were irreversibly lost because of graft reaction. The major complication was the formation of posterior subcapsular cataract, which occurred in approximately 32% of the patients. The final postkeratoplasty visual acuities were significantly improved. In the patients who did not develop a cataract, 86% achieved visual acuity of 6/12 (20/40) or better. In the patients who did develop cataracts, 68% achieved visual acuity of 6/12 (20/40) or better.

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VIRUS-SIMULATING STRUCTURES IN THE OPTIC NERVE HEAD IN CREUTZFELDT-JAKOB DISEASE

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Creutzfeldt-Jakob disease is a progressive, fatal, degenerative disease of the central nervous system in man which, because of its typical pathologic lesions and its transmissible nature, has been included in the classification of subacute spongiform virus encephalopathies[1] or slow virus infections together with Kuru in man, scrapie in sheep, and transmissible encephalopathy in mink. Creutzfeldt-Jakob disease was first transmitted to laboratory animals in 1968[2] by intracerebral injection of affected human brain and since has been transmitted with serial passages to a variety of species. Of particular interest to ophthalmologists was the report by Duffy and associates[3] of the possible transmission of Creutzfeldt-Jakob disease from person to person by corneal transplant and its experimental transmission in guinea pigs by intraocular injection of corneal material from infected animals.[4]

Virus-like particles have been found in brains in both natural and experimental Creutzfeldt-Jakob disease,[5-11] but there has been no attempt to identify such agents in the eye. We report herein the results of ultrastructural examination of ocular tissues from a patient with Creutzfeldt-Jakob disease with emphasis on identification of possible virus-like particles.

CASE REPORT

A 68-year-old man was well until September 1976, at which time his daughter noted that he seemed to be having fewer temper tantrums. The patient had been a heavy wine drinker, up to a half gallon per day, but in December 1975 had changed to occasional beers. He ate poorly while drinking. In October 1976, he had onset of vertigo, persisting for three weeks. The patient became ataxic and dysarthric, complaining of tinnitus, and his gait deteriorated with veering to the left. On Nov. 1, 1976, myoclonic jerks were observed during sleep, the patient was disoriented in his home, he was unable to work the water faucet, and had difficulty remembering recent events. Visual hallucinations were also observed by the patient. On Nov. 6, 1976, the patient was admitted here.

Results of the physical examination were unremarkable except for the neurologic examination. The patient was oriented to his name, knew the city, his birthdate, his age and address, but was unable to be more specific about recent events. Cranial nerve examination recorded vision as grossly normal; bilateral sixth nerve weakness was noted; up-gaze weakness and bilateral nystagmus on horizontal gaze was noted. Ophthalmoscopic examination was unremarkable. The rest of the cranial nerves were intact. Strength and tone were normal in the extremities. The sensory examination revealed loss of vibratory sense in both feet. Cerebellar testing showed marked dysmetria bilaterally with finger to nose testing. His gait was wide based, and the jaw-jerk was brisk. Plantar reflexes were normal.

Results of tests were unremarkable except for an eosinophilia of 7%, increased alkaline phosphatase of 225 units, and lactic acid dehydrogenase of 400 units. Cerebrospinal fluid on Nov. 16, 1976, revealed a protein of 50 mg/100 ml, glucose of 84%, and no cells. Skull x-rays were normal, and computed tomography scan was normal. Numerous electroencephalograms were performed, beginning on Nov. 8, 1976, and eventually showed deteriorating stereotyped periodic biphasic or triphasic discharges.

The patient's condition progressively deteriorated. On Nov. 16 1976, myoclonic jerks were detected. On Nov. 23, 1976 the patient was mute and his arms were rigid. He had intermittent conjugate deviation of the eyes to the left and right. Bilateral extensive

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plantar reflexes developed. Right-sided focal seizures were observed on Dec. 4, 1976, and cortical blindness was recorded on Dec. 23, 1976. The patient's condition continued to deteriorate and he died on Feb. 25, 1977.

MATERIAL AND METHODS

We removed the eyes at autopsy immediately after death and immersed the right globe in cold Karnovsky's fixative in 0.05M phosphate buffer. After hardening for 15 minutes, the eye was gross-sectioned as previously described[12]; gross examination was performed with the calottes immersed in fixative. Blocks of cornea, retina, and optic nerve head were cut with a razor blade and fixed overnight in cold phosphate-buffered Karnovsky's solution. The tissues were washed for several hours in a 0.05M phosphate buffer solution and postfixed for one hour in a 1% osmium tetroxide solution in the same buffer. They were washed in the buffer for 1½ hours, dehydrated in an ascending series of acetone solutions, and embedded in Spurr resin.

One micrometer sections were cut with an ultramicrotome and stained with toluidine blue. Ultrathin sections were collected on uncoated copper grids, stained with an alcoholic solution of uranyl acetate and lead citrate, and examined and photographed with an electron microscope. Tissue from the left eye was frozen in liquid nitrogen for transmissability studies.

RESULTS

General pathologic findings revealed marked cachexia and bronchopneumonia. On neuropathologic examination the brain weighed 1,025 g and externally showed only marked bifrontal cerebral cortical atrophy and occipital atrophy of moderate degree (Fig. 1). Coronal sections confirmed the frontal and occipital cortical atrophy. Cortex was tan and shrunken to less than 2 mm in width in many areas. Lateral ventricles were dilated, caudate nuclei were markedly atrophic, and substantia nigra was depigmented.

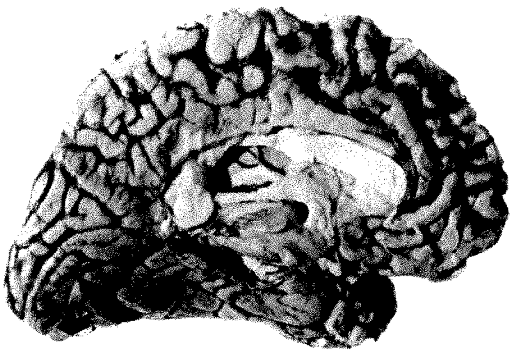

Fig. 1 (Roth and associates). Cortical atrophy present in frontal and occipital lobes (×0.6).

Microscopic findings included generalized neuronal loss in the cerebral cortex, striatum and substantia nigra, and was most severe in the frontal cortex. In many areas the cortical neurons had been entirely replaced by large gemistocytic astrocytes (Fig. 2). Status spongiosus was prominent in the deeper cortical laminae, particularly in areas of mild to moderate neuronal loss (Fig. 3). No inclusion bodies or inflammatory cells were found.

The right eye measured 24.8 mm in

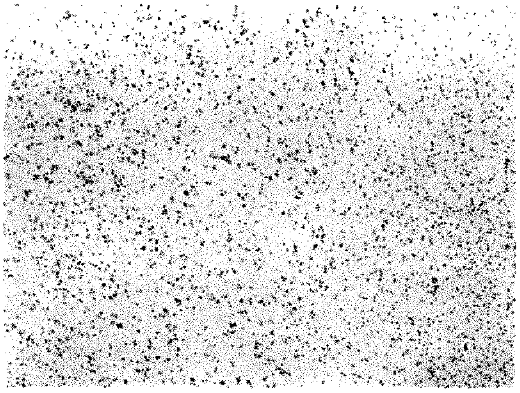

Fig. 2 (Roth and associates). Junction of cortex (left side of micrograph) and white matter (right side of micrograph) occipital lobe shows spongiform degeneration of neuropil most prominent in deep cortex. There is diffuse neuronal loss and marked astocytosis (hematoxylin and eosin, ×80).

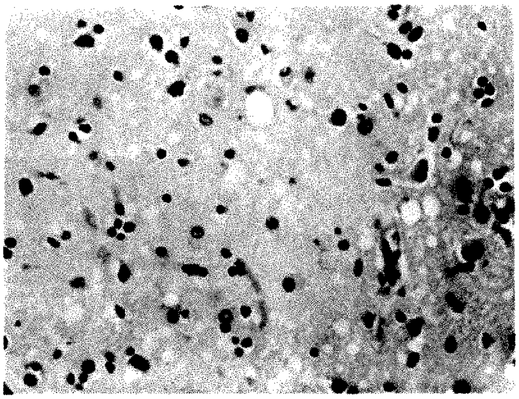

Fig. 3 (Roth and associates). Spongiform degeneration and gliosis in gray matter of occipital lobe (hematoxylin and eosin, ×320).

anteroposterior diameter. The cornea was translucent. The anterior segment and vitreous cavity were otherwise unremarkable. No retinal or macular lesions were seen grossly. The optic nerve head had a small conical cup. No gross optic nerve atrophy was apparent.

On light microscopy the cornea showed marked changes in its epithelium (Fig. 4). This was diffusely thinned with disorganization of its cells. Superficial cells were flattened and stained densely with toluidine blue. Epithelial basal lamina was intact and Bowman's layer, stroma, Descemet's membrane, and endothelium were unremarkable. The remainder of the ocular structures showed no significant pathologic changes. The macular area of the neurosensory retina and the optic nerve head were unaffected; the macular area showed no inner layer atrophy and the optic nerve head showed no evidence of nerve fiber atrophy, gliosis, or spongiosis (Fig. 5).

On electron microscopy, marked disorganization and degenerative changes in corneal epithelium were found; these included extreme osmiophilia, absence of cytoplasmic organelles, flattening of cell bodies and loss of nuclei in superficial layers and vacuolization of cytoplasm in basilar layers. The remainder of corneal structures were intact. There was no evidence of virus-like particles in corneal sections.

In the optic nerve head, however, we saw multiple particles within cell bodies and processes of many astroglia. These consisted of electron-dense cores surrounded by lucent zones and concentric membranous rings having an overall diameter of 125 to 200 μM (Fig. 6 and 7). Occasional pockets of these structures contained budding particles (Fig. 8). No particles were evident inside of nuclei.

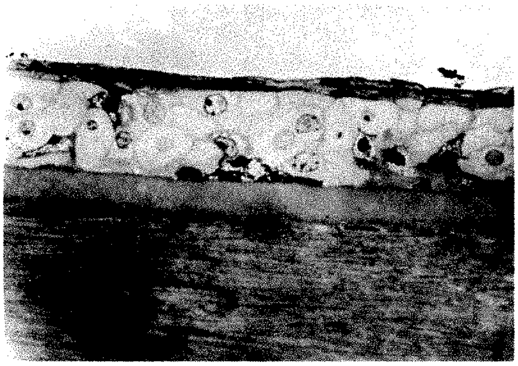

Fig. 4 (Roth and associates). Corneal epithelium is thinned with cellular disorganization. Its basal lamina, Bowman's layer, and superficial stroma are intact (toluidine blue, ×200).

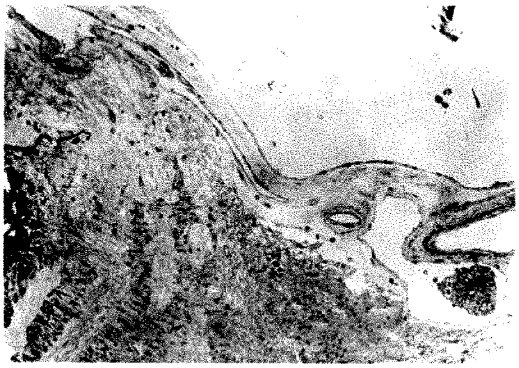

Fig. 5 (Roth and associates). No nerve fiber atrophy or gliosis in temporal aspect of optic nerve head (toluidine blue, ×80).

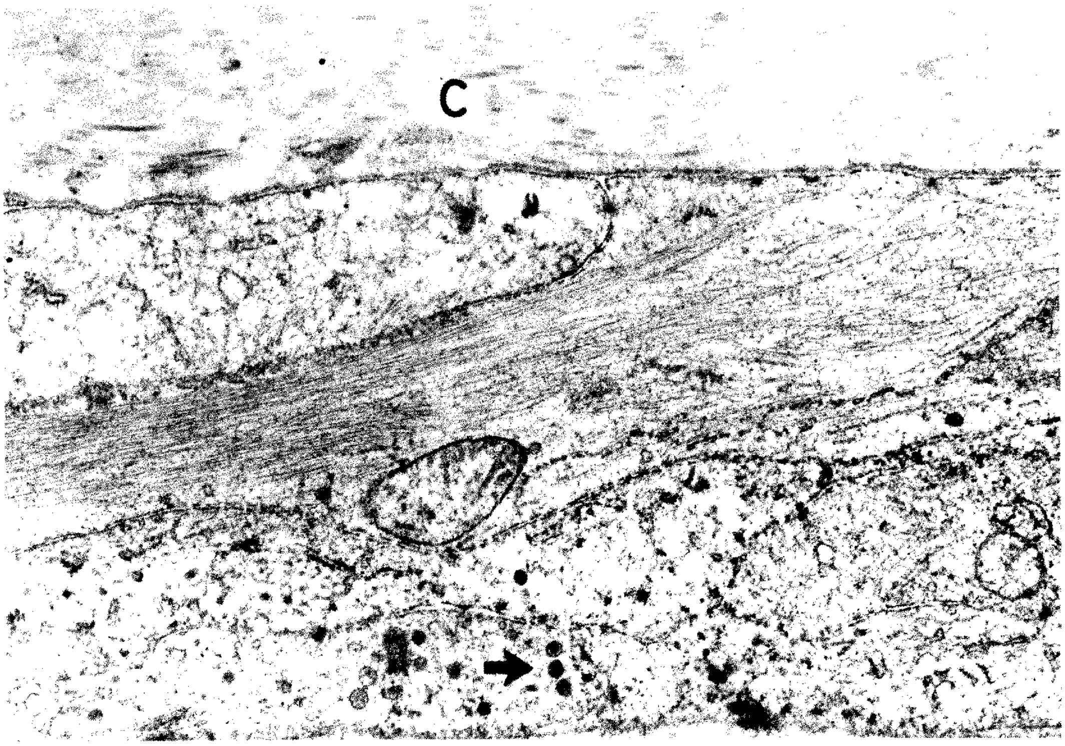

Fig. 6 (Roth and associates). Virus-like particles (arrow) in astroglial process of optic nerve head. C designates the collagen of central connective tissue meniscus (×4,800).

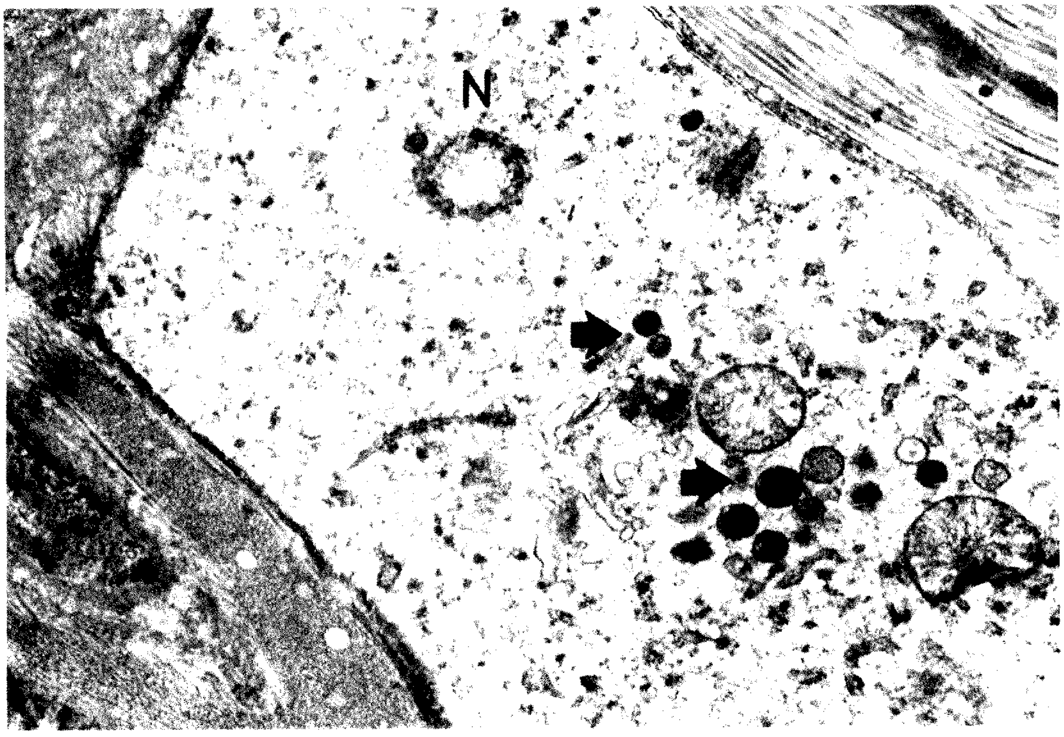

Fig. 7 (Roth and associates). Particles (arrows) in glial cell body have electron-dense core and concentric membranous rings. Tip of cell nucleus (N) contains pores that may simulate virions (×9,600).

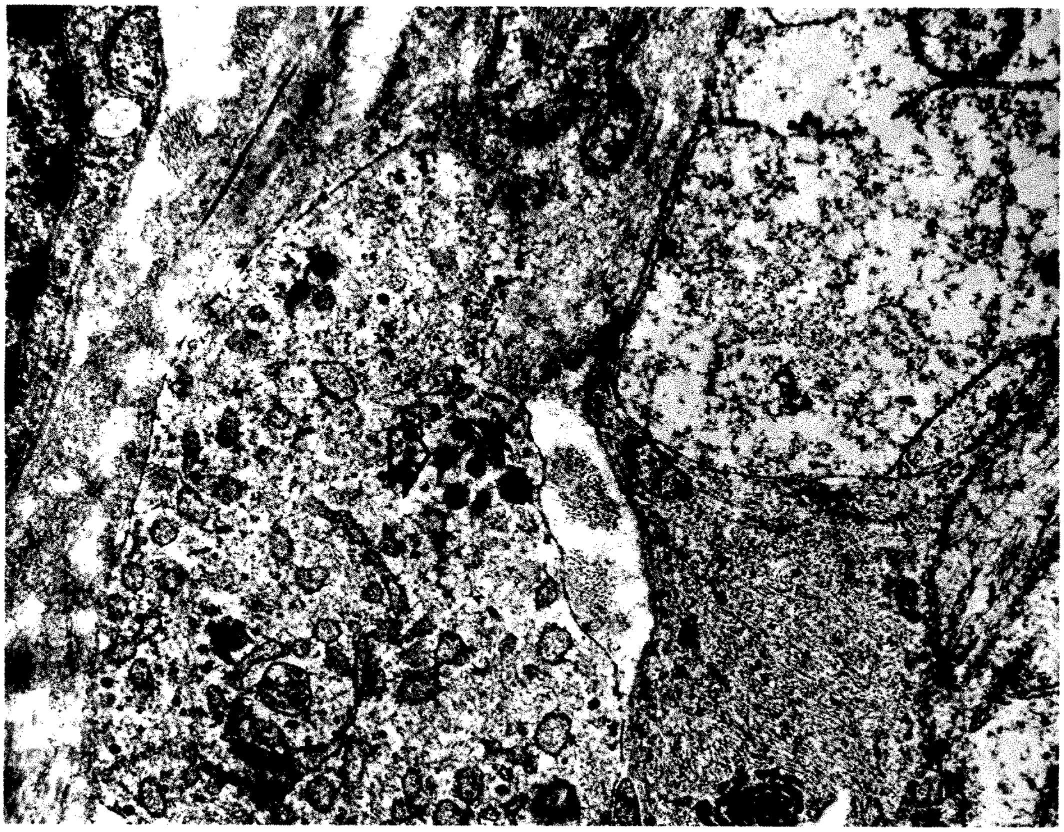

Fig. 8 (Roth and associates). Pocket of virus-like particles (arrow) appears to contain budding virions (×9,600).

The glial cells containing particles, as well as other glia and nerve fibers, showed no other ultrastructural changes.

DISCUSSION

The apparent transmission of Creutzfeldt-Jakob disease between humans by corneal transplantation[3] implicates the presence of the infective agent in the eyes of affected patients. Herzberg and associates[13] suggested that the high incidence of the disease in Israelis of Libyan background was related to their practice of eating sheep eyeballs. The presence of an infectious agent in the eye is supported by experimental infection of guinea pigs by inoculation of corneal tissue from affected animals into the anterior chambers of healthy specimens.[4] It is also of interest that transmissible mink encephalopathy has been transmitted in hamsters by intracerebral injection of corneal epithelium.[14] In the same study, neither cultured corneal epithelium nor aqueous humor was similarly infective. The difference between original and cultured epithelial infectivity was explained either by failure of infected epithelial cells to replicate in tissue culture or, more probably, that infectivity is associated with the free nerve fibers in corneal epithelium.

We examined the ocular tissues from our patient specifically for the presence of an infective agent. The corneal changes we saw were nonspecific and probably related to the marked terminal morbidity of the patient. We found no morphologic evidence of virus-like particles in corneal tissue. However, we were able to identify only a few nerve endings in the altered epithelium and, if Marsh and Hanson's[14] hypothesis that infectivity is associated with the nerves is correct, this might account for the negative finding. We did identify numerous virus-like particles within glial cells of the optic nerve head. These structures were morphologically and dimensionally similar to those reported in the numerous reports describing virus-like particles in brain tissue of patients with Creutzfeld-Jakob disease.[5-11] However, the significance of these particles in the pathogenesis of Creutzfeldt-Jakob disease is questionable.[15] The reports may have shown virions that were incidentally present in the patient but had no relationship to the disease in question. Rogers and associates[16] described in chimpanzees with Kuru multiple latent viruses unassociated with the disease. Many regularly occurring cell components and other unidentifiable structures may mimic viruses when viewed by the electron microscope.[17] We believe the particles we have described are such virion-simulating structures and it is probable that many of the particles previously reported are similar.

If the structures reported as virus-like are not the causative agents of Creutzfeldt-Jakob disease, why have these not yet been found? They are atypical viruses which, although filterable, do not elicit host immune mechanisms, do not evoke viral-like inflammatory response in brain tissue, and possess physical properties unlike other viruses.[18] They show marked resistance to inactivation by many conventional methods including heat, ultraviolet radiation, and formaldehyde; this has led to recommendations of extreme caution in evaluation of patients having the disease.[19-23] Studies suggest that the virus might be small naked nucleic acids bound to membranes,[18] perhaps similar to the viroids causing plant diseases,[24] and could activate latent viruses in the host.[25] This would explain many of its atypical properties and the failure to identify it by the electron microscope.

The blindness in patients with Creutzfeldt-Jakob disease has been classically described as cortical in nature. No clinical ocular changes have been reported in such patients. Lesser and associates[26] found histologic atrophy of the optic nerve and the inner retinal layers at autopsy in a patient who showed no optic atrophy clinically, although visual loss was present during life. The authors were unable to explain this disparity. Barnett and Palmer[27] and Buyukmihci and associates[28] described histologic degenerative changes in the outer retinal layers of animals infected with scrapie. Buyukmihci and associates[28] additionally found vacuoles in the optic nerves. We found no evidence of retinal or optic atrophy either clinically or pathologically in our patient, and the severe occipital degeneration accounted for his visual loss.

The findings in our case indicate that there are no specific pathologic changes in the eyes of patients with Creutzfeldt-Jakob disease. The axons and glial elements in the nerve head, in the anterior optic nerve, and in the retina were structurally intact. No evidence for an infectious agent was found in any portion of the eye.

SUMMARY

A 68-year-old man was treated for and died of Creutzfeldt-Jakob disease. At autopsy we found multiple virus-like particles in the optic nerve head, but saw no similar structures in the cornea. Although these particles were morphologically similar to those previously reported in brain, we believe that they are not virions but unrelated cellular structures. We speculate that the causative agents may be

naked membrane bound nucleic acids rather than true viruses. We found no optic atrophy or other specific pathologic changes in the eyes; severe occipital cortical degeneration was responsible for the patient's visual loss.

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NOTES, CASES AND INSTRUMENTS

ACUTE MYOCARDIAL INFARCTION AFTER FLUORESCEIN ANGIOGRAPHY

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Myocardial infarction occurring after fluorescein angiography was suggested by several investigators[1-4] but this complication has only recently been documented.[5] We describe herein a case of acute myocardial infarction occurring after fluorescein angiography. The stress of the examination and of the photography may have contributed to the patient's complication.

CASE REPORT

A 68-year-old man was examined here in June 1976. He had a two-year history of decreased vision in the right eye. Visual acuity was counting fingers in the right eye, and 6/9 (20/30) in the left eye. His right macula had no foveal reflex, but had various size drusen with areas of clumping and atrophy of the pigment background. The left eye had only scattered drusen in the macula. Visual field testing with the Goldmann perimeter showed a relative central scotoma in the right eye; the left visual field was normal. The patient was scheduled for fluorescein angiography to evaluate the right macular changes.

The patient had a history of vascular disease including an inferior myocardial infarction in 1973, a right femoral endarterectomy for symptoms of claudication in 1969, and the onset of claudication in his left leg in 1974.

Dilation of the right pupil was done approximately 30 minutes before angiography by using two instillations, five minutes apart, of both 10% phenylephrine and 1% cyclopentolate. The fluorescein angiogram was performed. There was mottled hyperfluorescence in the right macula in areas of retinal pigment epithelial atrophy without evidence of hemorrhage, neovascularization, or leakage. The findings were interpreted as compatible with senile macular degeneration.

The patient complained of sudden, sharp pains in his chest radiating to his left arm 30 minutes after the injection of fluorescein. He developed dyspnea and diaphoresis without nausea or vomiting. An electrocardiogram was taken immediately and showed an old inferior myocardial infarction, but comparison with previous tracings revealed additional ST wave depression in leads II and av F with T waves peaked in leads V3 to V5. Nitroglycerin sublingually failed to relieve the pain and the patient was admitted to the coronary care unit. His blood pressure was 110/74 mm Hg; he had a regular pulse of 80 beats per minute, and a respiratory rate of 16. Rales were present at the left lung base; no cardiac gallop or murmur was detected.

Laboratory findings showed the following: leukocyte count, 9,500/mm^3; hemoglobin, 15.1 g/100 ml; blood urea nitrogen, 11 mg/100 ml; serum electrolytes, normal; and glucose, 135 mg/100 ml. Chest x-ray did not show congestive heart failure.

Serial electrocardiograms over the next five days showed the development of QS deflections in leads V1 through V4, and the inversion of T waves in leads V2 to V5. Serum creatine phosphokinase on admission was 100 units (normal, 50 to 180 IU/l), 1990 units on the next hospital day, and 81 units on the fifth hospital day. Lactic dehydrogenase was 156 units on admission (normal, 90 to 210 IU/l), 530 units the next day, and 238 units on the fifth day. Serum glutamic oxaloacetic transaminase was 37 units on admission (normal, 8 to 30 IU/l), 273 units the next day, and 34 units on the fifth day. The cardiac enzymes and electrocardiographic changes were interpreted as consistent with acute anteroseptal myocardial infarction.

The patient had no chest pain after the fifth day; he was discharged after two weeks. During the 30 months after the myocardial infarction, the patient has had no recurrence of chest pain. He has had increasing difficulty with his left leg claudication. Visual acuity has remained unchanged.

DISCUSSION

The sudden radiating chest pain, diaphoresis, and dyspnea associated with the rise in cardiac enzymes and serial changes in the electrocardiograms established

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the diagnosis of acute myocardial infarction occurring after fluorescein angiography in this case. It was not possible to determine whether the myocardial infarction was causally or coincidentally related to the injection of fluorescein.

The patient had no nausea, vomiting, or bradycardia to suggest a vasovagal hypotensive episode as the cause. There was no reason to suspect spasm of the coronary arteries in a patient with diffuse peripheral vascular disease and a history of a previous myocardial infarction. No urticaria or itching occurred to indicate an allergic response. Transient systemic effects of the topically applied phenylephrine dilating drops[6] could have increased demand on the heart, even though the vital signs were normal when taken over an hour after instillation of phenylephrine. His diseased myocardium would have been more vulnerable to a possible hypotensive effect of rapidly lowering the carbon dioxide[7] through hyperventillation from the stress of the situation.

Patients with cardiovascular disease would probably be the most susceptible to stress from angiography. The only other documented case of myocardial infarction after fluorescein angiography[5] occurred in a patient with hypertension and severe peripheral vascular disease. Another case of apparent myocardial infarction after angiography[1] occurred in a patient with a history of two previous myocardial infarctions. Other reports[1-4] have not provided details for comparison of common factors.

Two groups[8,9] have performed electrocardiographic monitoring of their patients undergoing fluorescein angiography. Although they failed to find significant changes with electrocardiographic studies, only a few of their patients had myocardial or peripheral vascular disease.

We do not consider fluorescein angiography to be contraindicated in patients with cardiovascular disease, but we do consider the patient's general health before performing angiography. We take the precautions of assessing cardiac rhythm by palpating the pulse and checking the blood pressure before injection of fluorescein. Drugs and equipment for cardiopulmonary resuscitation are immediately available. Most importantly, we evaluate every reaction after fluorescein angiography.

SUMMARY

A 68-year-old man with a history of previous myocardial infarction and peripheral vascular disease had an acute myocardial infarction 30 minutes after the injection of intravenous fluorescein for fundus angiography. The situational stress or phenylephrine dilating drops could have contributed to the complication.

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VITREORETINAL DISSECTION INSTRUMENTS

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Dissection of tissue planes in the vitreous cavity, such as separation of cortical vitreous and other epiretinal tissue from the inner surface of the retina, is an important part of modern vitreoretinal surgery techniques in some cases.[1-5] These manipulations are usually performed with a bent-tip needle[1] or with a special vitreoretinal pick that includes a fiberoptic light source designed to be used with the O'Malley Ocutome system.[6] Vitreoretinal picks and probes with different tip designs are desirable to engage and manipulate membranes and tissue bridges having different physical properties, configurations, and orientations. Together with the Mentor Divison of Codman-Shurtleff, Inc., we developed and evaluated a series of special vitreoretinal picks designed to facilitate these delicate manipulations and provide different tip designs for various situations.*

MATERIAL AND METHODS

Three similar vitreoretinal dissection instruments have been developed, differing only in design of the tip. Each instrument weighs approximately 6.3 g and is 151 mm (6 inches) in length, including a 100-mm (4-inch) handle portion that is 4 mm in diameter and has a fine diamond-knurl surface pattern to facilitate easy grasping. The intraocular portion of the instrument has a constant diameter of 0.9 mm except for the most distal 7 to 10 mm, which is tapered somewhat toward the angulated tip. The three tip designs include: (1) a hook bent at an angle of 90 degrees; (2) a hook bent at a 130-degree angle; and (3) a blunt-tip right-angle spatula (Fig. 1). The ends of the two hooked instruments are not sharp, and the overall diameter of the hooked ends is the same as the constant-diameter, intraocular portion of the shaft. The diameter of the right-angle spatula tip is 1.5 mm.

Each instrument is manufactured from an alloy of titanium (Mentanium). This alloy is corrosion resistant, stronger than stainless steel by weight, and 43% lighter than stainless steel. The instruments have a special anodized finish that is blue-gray in color, and this finish minimizes glare caused by reflected light from the fiberoptic intraocular illumination probe. Each instrument has a flat portion near the distal end of the handle that contains an identifying number, and the instruments are stored in a special metal case that protects the delicate tips during storage, sterilization, and when not in use during surgery (Fig. 2).

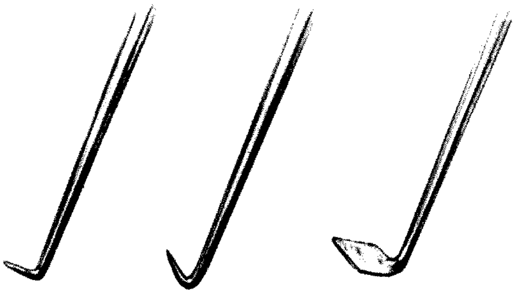

Fig. 1 (Michels, Rice, and Ober). Three tip designs for the vitreoretinal dissection including: (1) a hooked tip bent at a 90-degree angle, (2) a hooked tip bent at a 130-degree angle, and (3) a blunt-tip right-angle spatula.

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*Available from Mentor Division, Codman and Shurtleff, Inc., Randolph, MA 02368.

From the Department of Ophthalmology, Johns Hopkins University School of Medicine, Baltimore, Maryland (Drs. Michels and Rice), and the Department of Ophthalmology, University of Southern California, Los Angeles, California (Dr. Ober).

Reprint requests to Ronald G. Michels, M.D., Wilmer Institute, Room 116, Johns Hopkins Hospital, Baltimore, MD 21205.

DISCUSSION

These vitreoretinal instruments were developed to facilitate delicate intraocular tissue dissections, including separation of epiretinal membranes from the inner surface of the retina. They are easy to handle, lightweight, and glare resistant; and the tips are designed to engage and manipulate tissue from various directions and in a variety of circumstances. For example, the 90-degree-hooked-tip instrument is used to engage the proximal edge of membranes and the 130-degree-hooked-tip instrument is used to engage the distal edge when the dissection force must be directed toward the quadrant through which the instrument is introduced. The blunt-tip right-angle spatula is used to elevate vitreous and other tissue bridging between one area of retina and another, and to break delicate vitreoretinal adhesions by side-to-side movement.

We have used these vitreoretinal dissection instruments together with the O'Malley Ocutome system in more than 75 vitrectomy operations during the last six months. These instruments are used interchangeably with the Ocutome cutting probe, vacuum-cleaning needle, and intraocular scissors through the same 20-gauge sclerotomy, and illumination is provided by a separate fiberoptic probe. However, these instruments can also be used with a full-function vitrectomy probe containing the features of a cutting mechanism, suction, infusion, and illumination systems in a single tip introduced through a separate pars plana sclerotomy.

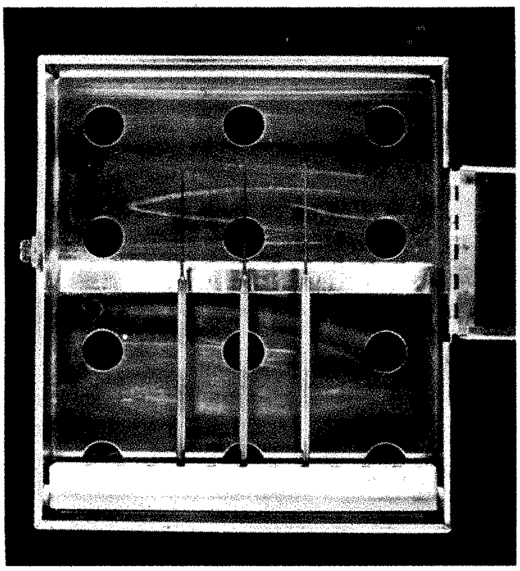

Fig. 2 (Michels, Rice, and Ober). Special metal case for storage of vitreoretinal dissection instruments. Each instrument is identified by number, and hinged top protects the delicate tips during sterilization and storage. Large holes in the metal case assist during ethylene oxide sterilization.

SUMMARY

We developed and evaluated clinically a series of three vitreoretinal dissection instruments with a constant-diameter shaft and various tip designs including a 90-degree hooked tip, a 130-degree hooked tip, and a blunt-tip right-angle spatula. The instruments, manufactured from a titanium alloy, are lightweight and glare resistant. We have used these instruments interchangeably with other vitreous surgery instruments introduced through a 20-gauge sclerotomy. They facilitate delicate manipulations associated with separation of epiretinal membranes from the inner retinal surface.

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MEETINGS, CONFERENCES, SYMPOSIA

EDITED BY THOMAS CHALKLEY, M.D.

ASSOCIATION OF UNIVERSITY PROFESSORS OF OPHTHALMOLOGY: 12TH ANNUAL MEETING

The 12th Annual Meeting of the Association of University Professors of Ophthalmology (AUPO) in January 1979 was marked by three noteworthy features. First, it was the first meeting of the AUPO attended by the directors of non-university ophthalmology resident training programs. Thus, about 60% of the approved residency programs in the United States were represented. Second, the remarkably successful first residency match program for ophthalmology was described in detail. Third, the governmental impact upon residency training programs was discussed from three vantage points: (1) research—Carl Kupfer, Director of the National Eye Institute, cautioned us about potential lean research support ahead; (2) Veterans Administration situation—John H. Mather of the Veterans Administration's Central Office explained his views, which made all those who were present concerned about the optometry-ophthalmology interface in the Veterans Administration; and (3) the keynote speaker, Leonard W. Cronkhite, President of the Medical College of Wisconsin, whose observations of the government's increased control of medical schools and university hospital staffs stimulated all to be concerned about the opportunities for extending specialty training in the future.

The meeting of the AUPO this year could be considered a comeback. Only last year, one of the founding fathers, A. Edward Maumenee, warned the organization that its past role in the attainment of the National Eye Institute was commendable, but no longer suffices as a continuing goal of the AUPO. The reports of activity fostered by the AUPO since last year are noteworthy:

1. The matching program organized under the auspices of the AUPO by August Colenbrander was done without a single error (and without a computer). The trustees of the AUPO had contacted each ophthalmology program director in the United States to attain the 90% participation necessary for the success of the venture. The statistics showed that 575 qualified applicants completed the forms, interviews, and listed their preferred programs and 459 positions were available to these applicants. Four hundred fifteen matched and the remaining 116 were available for those positions to be filled with "late bloomers" or secondary matching efforts. Needless to say, the members voted overwhelmingly to continue the matching program for another year.

2. The manpower report[1] was published last year and the AUPO contributed greatly to this major first step in looking at the critical issue of trying to see what the future needs of the public may be for ophthalmologists. Data for Parts 2, 3, and 4 are being sought now by a committee of the AUPO.

3. The input to the Veterans Administration from the AUPO has gradually increased over the past year and anecdotal accounts of problems in the Veteran Administration strongly suggest that the AUPO should have greater input into the Veterans Administration administrative system since at least 50% of all eye residency training programs rely on V. A. hospitals for at least a portion of their residents' training.

4. The financial structure of Departments of Ophthalmology are being threatened by new legislation which may make appropriate payments for ophthalmologists' services in university hospitals almost nonexistent. Revision of these proposed regulations is being sought through the AUPO's relationship to the AAMC.

5. The importance of the internship is being rediscovered and through the American Board of Ophthalmology's decision to require such a year in the future, the AUPO must interact with AAMC and other agencies to assure that appropriate first-year postgraduate positions (now called the transition year) are available for those accepted for a residency in ophthalmology.

Only 14 years ago a small group of leaders in ophthalmology[2]—A. Edward Maumenee, John M. McLean, Bernard Becker, Frank W. Newell, Michael J. Hogan, and David G. Cogan—conceived the idea that AUPO should be formed. Those strong individuals assured success of the organization during its formative years. It is hard to conceive of any organization faltering with people such as these in it. Now as those leaders pass the reins to others, the real question as to the strength and viability of the organization arises. At this moment, those of us who attended the meeting this year, ably chaired by the 1978 AUPO president, Richard Schultz, were impressed with the real need for such an organization. The members further noted the importance of their guests, the non-university program directors, and are changing the rules to admit such members. The strong heritage from our leaders in ophthalmology includes the AUPO, which appears likely to continue to be a viable force.

ROBERT D. REINECKE

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Published monthly by the Ophthalmic Publishing Company
Tribune Tower, Suite 1415, 435 North Michigan Avenue, Chicago, Illinois 60611

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A CRISIS IN THE MANAGEMENT OF PATIENTS WITH CHOROIDAL MELANOMA

In recent years much of the writing concerning choroidal melanomas has been related to problems in diagnosis and use of new techniques to improve our diagnostic abilities. These include such techniques as fluorescein angiography, radioactive phosphorus (^{32}P) uptake studies, and ultrasonography. Studies published by Ferry[1] and Shields and Zimmerman[2] have indicated an approximate 20% error in the diagnosis of eyes contributed to the Armed Forces Institute of Pathology. Many good clinicians in the country have almost no problems in differentiating the group of nevi—melanoma lesions from other intraocular lesions—which in the past have been confused with melanomas. It has been several years since our eye pathology laboratory at Baylor has received an eye enucleated because of a suspected melanoma which contained some other type of lesion. Approximately ten years ago one of our former residents reviewed 100 consecutive cases of eyes enucleated because of suspected melanomas and found only two eyes that did not contain melanomas.

Now that we are able, for the most part, to differentiate the nevi-melanoma group of lesions from other simulating lesions our attention has now turned to management. In their persuasive study, Drs. Zimmerman and McLean go to great lengths with an extensive bibliographic review to make the point that enucleation may adversely affect the prognosis in patients with uveal melanomas. This hypothesis may prove to be correct but good evidence is not yet available to support this view. There are several points of disagreement and one concerns a statement regarding growth of nevi. Zimmerman and McLean make the statement that nevi may show evidence of growth but I am not personally convinced that nevi of the skin, conjunctiva, or uveal tract may grow. They certainly may acquire pigment or, in the conjunctiva, cystic changes. These changes may alter the appearance and often these changes occur at puberty and during pregnancy. Because a lesion of the choroid has been documented to enlarge very slowly photographically does not mean that the lesion in question is a nevus. It may eventually prove to be a slow growing malignant melanoma. I would agree with Zimmerman and McLean, however, that we continue to have a problem in differentiating large nevi from small melanomas. This is not only a problem in clinical differentiation but also it continues to be a problem differentiating choroidal nevi from spindle A melanomas histologically. Zimmerman and McLean reviewed 105 old cases of spindle A melanomas on file in the Registry of Ophthalmic Pathology and reclassified 15 of them as spindle cell nevi. Many ophthalmic pathologists have difficulties in differentiating these lesions and would not agree with their interpretations.

One of the main problems with the authors' data is that patients on whom the statistics are based are ones who have come to enucleation because they have been symptomatic. These are patients who have developed retinal detachments, macular changes, uveitis, secondary glaucoma, cataracts, vitreous hemorrhage, and the like. All of these are changes that indicate a lesion in a phase of accelerated growth or cytologically active. If one were to take a series of asymptomatic lesions that were picked up on routine examination, I do not think the death rate in these cases would rise to the 8% level in the first year after enucleation as indicated by the authors. Because a lesion is cytologically active and symptomatic would make it more susceptible to metastases within a period of several years after manifestation and enucleation. We need additional control studies to determine if the hypotheses proposed by Zimmerman and McLean are correct.

From a practical point of view publication of the study will stimulate much needed research in the field but for the time being will make it more difficult for the practicing ophthalmologist to deal with the individual patient. The patient with a large melanoma associated with a secondary retinal detachment and loss of useful vision would still be a candidate for enucleation preferably using some of the techniques recommended by Fraunfelder and associates.[3,4] The performance of a ^{32}P uptake study as a separate procedure by a consultant in these cases is not recommended because the patient is exposed to two surgical procedures, which might help to disseminate tumor cells. If a ^{32}P test is done in such a situation it should only be done at the time of planned enucleation so that the patient is exposed to only one surgical procedure and a minimum of manipulation. In other situations where there is an enlarging lesion of the choroid in a young patient with good health and useful vision, I recommend a combination of radiation therapy and photocoagulation as we have recently described.[5] If an enlarging lesion involves the peripheral choroid and ciliary body some of these patients can be treated by local resection after preliminary cryocoagulation or photocoagulation.

MILTON BONIUK

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A MORATORIUM ON ENUCLEATION FOR CHOROIDAL MELANOMA?

In their magisterial review of the role of enucleation in the management of choroidal and ciliary body melanomas, Zimmerman and McLean have concluded that an imposing body of data exists, much of it disconcertingly circumstantial and anecdotal, that enucleation is ineffective in saving lives. They believe that posterior uveal melanomas metastasize late when left alone, but that an increased rate of fatality in the second postoperative year is in part attributable to events (surgical or immunologic, or both) surrounding the act of enucleation.

In my view they have made the most cogent argument they can with the imperfect clinical and statistical information on the biologic behavior of choroidal melanoma presently available. To test my own reaction to their construction of the case against enucleation, I asked a group of six professional peers, all but one outside of the mainstream of clinical ophthalmology, to offer a critique of the paper: an experienced clinician with an orientation toward ophthalmic pathology (Paul Henkind); an ocular immunologist (Julian Manski); a general surgeon with experience in performing "no-touch" abdominal tumor surgery (Frederick Herter); a dermatopathologist with an interest in cutaneous melanoma (David Silvers); a general surgical pathologist (Karl Perzin); and a cellular pathologist (Gabriel Godman). They all found the essential clinical features of Zimmerman and McLean's thesis to be compelling, although they expressed reservations about some of the explanations for the clinical observations.

Despite the seductive plausibility of Zimmerman and McLean's argument—a virtuoso performance demonstrating the bias of perspective that enters into the handling of any set of moldable data—a nagging concern remains that their conclusion has not been proved beyond a reasonable doubt. Have they fallen prey to the logical fallacy of post hoc, ergo propter hoc, the error of arguing from a temporal sequence to a causal relationship? Is it possible that when a choroidal melanoma has reached a size necessitating its removal its biological character has also undergone a radical transformation with the concomitant production of occult metastases undetectable by current methods of noninvasive investigation? One could take as time zero the surgical removal of any gastrointestinal or renal tumor, and say: "The tumor was removed and the patient died two years later—just look at what your surgery has done."

Although the previous example may be facetious, the parallelism between cutaneous melanoma and choroidal melanoma is both awkward and worrisome. Wallace Clark and Matthew Harris, two well know specialists in cutaneous melanoma, have seen only a few patients with untreated primary cutaneous melanomas (apart from neglected fungating tumors, which would be the cutaneous counterpart of an intraocular melanoma with proptosis) who had clinically disseminated disease at the time they were first seen. Of those patients who have a fatal cutaneous melanoma, 40% will be dead within two years after the excision of their tumors. These features are remarkably reminiscent of what happens in the clinical course of enucleated choroidal melanomas, yet no dermatologist believes that the excision of skin tumors causes metastases. A dermatologic report[1] shows that an incisional biopsy through a cutaneous melanoma, during which malignant cells have to be dislocated into vascular or lymphatic channels, does not worsen the prognosis.

The intellectual and clinical melee that will follow in the wake of the article by Zimmerman and McLean should not obscure the reality that a uveal melanoma showing progressive growth, if left untreated, will cause death after a variably prolonged period. Of Raivio's five patients and Sobanski's ten patients who refused enucleation for their choroidal melanomas, 14 died from their disease, eight of these one to three years after the clinical diagnosis of their tumors.

If routine enucleation is deleterious to the survival of patients harboring "large" choroidal tumors, Zimmerman and McLean are willing to concede that enucleation probably does not adversely affect the outcome of "small" melanomas (tumors measuring 10 mm or less in greatest diameter and 2 mm or less in height). With our improved clinical and pathologic knowledge regarding the indistinct zone of classification between nevi and small melanomas, it may well be that the comparatively favorable outcome of many of these tumors after enucleation reflects their benign condition at the time of enucleation. Alternatively, some of these small tumors may be biologically malignant (small choroidal tumors occasionally contain epithelioid cells), but enucleation aborts their continued growth toward a fatal "critical mass." In cutaneous melanomas, the removal of a tumor with a thickness of 1.5 mm or less results in an 80% cure rate; if untreated, the majority of these tumors will inexorably grow to a thickness of 3.0 mm, which results in a 75% death rate.[2]

Experimental evidence in support of Zimmerman and McLean's interpretation that enucleation dislodges metastasizable clumps of tumor cells into the circulation ("intravasation") shows that, the larger the size of the tumor cell emboli, the more likely the cells are to implant successfully in the new micro-environment.[3] Although a few individual tumor cells are probably constantly seeding the bloodstream from the primary site, such small numbers of cells are usually destroyed by the host's defense mechanisms. In an animal study, only 1% of intravenously injected tumor cells survived after 24 hours, whereas less than 0.1% of cells had survived by the 14th day.[4] Enucleation probably creates larger cellular aggregates with enhanced capacity to survive in metastasis; nonetheless, the presence of cells in the blood should not be equated with the production of metastases.

While marshalling evidence to prove that enucleation is a culprit in promoting metastases from large intraocular melanomas, Zimmerman and McLean also indicate that such tumors are apt to contain more malignant cell types (particularly epithelioid cells). Regrettably cytomorphology is a weak reflector of the biologic properties of tumor cells, because tumors of identical histologic appearance may well be composed of cells with significantly different membrane components and other properties conducive to metastasis. For example, there are benign cutaneous nevi composed at times almost exclusively of epithelioid cells; if such a lesion were transposed to the uvea it would precipitate a malignant diagnosis. Indeed, benign epithelioid cell nevi of the iris have recently been identified.[5] Although Zimmerman and McLean point out that there is a dearth of clinical information on what happens to small choroidal melanomas after long-term follow up, we have almost no information available on the clonal composition of these melanomas undergoing evolution and evading immunological defenses.

The emphasis on the mechanical factor in Zimmerman and McLean's study, therefore, sidesteps an even more important aspect of the metastatic phenomenon, that is, the intrinsic biologic properties of the tumor cells. What are the stages by which a metastasis is produced? With respect to intraocular melanomas, the tumor cells must invade blood cells (there are no intraocular lymphatic channels), and detach into the bloodstream. Thin-walled venules are the presumed pathway into the circulation for most solid tumors; as Zimmerman and McLean have pointed out, large intraocular melanomas abound in delicate dilated vascular spaces into which tumor cells can be pushed during an enucleation, that is, through surgical manipulations that are the ocular equivalent of a Valsalva maneuver. In the bloodstream tumor cells may aggregate together, reaching a sufficiently large size to metastasize successfully by becoming "hung-up" in the microcirculation. After extending outside of the capillaries, the ability of the microemboli to survive in the metastatic extravascular environment is brought about by a tumor angiogenesis factor. This substance, which is elaborated by the tumor cells, promotes the proliferation of adjacent endothelial cells causing vascularization of the tumor seedlet.

The precondition for metastasis is that the tumor cells have the requisite biologic properties to survive in distant sites. Ex-hypothesi, if the cells of a benign spindle cell nevus are trypsinized and directly injected into the veins of a "human volunteer" none of the cells should survive in the form of a metastasis. Conversely, if the cells of a malignant melanoma are trypsinized and injected intravenously, one would discover that some of the cells do produce metastases, depending on the immune status of the host.

The best available experimental animal data suggest that there are heterogeneous subpopulations of tumor cells in the primary lesion, and that the ability of tumor cells to metastasize is a pre-existent property resident in limited tumor cell lines, rather than being representative of a random adaptation of tumor cells to survive once ensconced in a favorable milieu.[6] Extrapolating these findings to the growth of a uveal melanoma, clones of tumor cells that escape immunologic surveillance may emerge with the ability to metastasize. Enucleation would therefore accelerate the formation of metastases by the early dislodgement into the bloodstream of metastasizable subpopulations of cells within the tumor, but these cells would probably have invaded the bloodstream on their own at a later date, if indeed they had not already done so before enucleation.

The in vivo immunologic backdrop of the choroidal melanoma story is also more intimidatingly complex than limned by Zimmerman and McLean. By the time uveal melanoma cells have become malignantly transformed with acquisition of intrinsic cellular properties for metastasis, they have also acquired a set of cell surface neo-antigens that should be capable of inciting immunocompetent cells to reject them. Unfortunately, the immune system may paradoxically react in some instances to enhance tumor cell growth. Antigens shed by the tumor cells, blocking antibodies, antibody fragments, and immune complexes can all apparently interfere with the cancer immunosurveillance system. We are now confronted with an immune system that functions differently from person to person, depending on their variable histocompatibility and genetic patterns, and that involves the idiosyncratic orchestration of B-lymphocytes, helper T-cells, killer T-cells, blocking antibodies, cytophilic antibodies, opsonizing antibodies, primed macrophages, and suppressor T-lymphocytes, inter alia, all acting in concert upon the peculiar features of the neoplasm.

The suppressor T-cell function is particularly fascinating.[7] A subset of T-lymphocytes called suppressor cells works to turn off immunoreactions by directly interacting with other T- or B-cells to prevent excessive responses and autoimmune processes. In some cancerous conditions, much of the host's inability to cope with a tumor may be traceable to the disadvantageous effects of suppressor T-cells on other lymphoid elements, which could eradicate the tumor if not interfered with. In some animal tumor models, suppressor T-cells have been selectively depleted by total body irradiation, splenectomy, or thymectomy which enables the remaining killer T-lymphocytes to attack and retard progression of the tumor. Drugs such as cyclophosphamide, colchicine, indomethacin, and cortisone also have the desirable effect of selectively depleting suppressor T-cells to the relative advantage of the effector cytotoxic T-lymphocytes.

Immunotherapy with BCG has not been successful in the treatment of cutaneous melanoma and other malignancies, and immunization of patients with preparations from their own tumors has sometimes shortened survival, which may result because nonselective immunostimulation activates as many or more suppressor T-lymphocytes as effector killer T-lymphocytes. In whatever manner the immune system plays upon the growth and modifies the metastatic potential of choroidal melanomas, enucleation itself must be an epiphenomenon to the "war of the cells."

One cannot dismiss the arguments presented by Zimmerman and McLean because of cavils over the manifest defects of some of the data they use; the same data are equally frail in justifying traditional enucleation. If they are wrong in alleging the enucleation causes metastases, they have nonetheless disabused us of our complacent attitude that enucleation as presently performed is an effective form of life saving therapy. Fortunately, guidelines are emerging for the management of choroidal melanomas. A consensus has been reached that small stationary tumors need not be enucleated. Slowly enlarging, asymptomatic tumors can probably also be prudently observed, but at shorter intervals. A dilemma arises when the tumor approaches a size where conservative local therapies (iridocyclectomy, eye-wall resection, cobalt plaque, proton beam irradiation, photocoagulation, or cryotherapy) may no longer be serviceable. It is in this uncertain area between doing nothing and doing something less drastic than enucleation that better indications will have to be developed. In this regard, the results of studies on survival after enucleation may not be valid with respect to subtotal ocular resections, because reported survivals have been excellent to date (possibly the result of interrupting the trajectory of smaller tumors toward malignant clones).[8,9] Finally, what is to be done for tumors that can be removed only by enucleation if they are to be treated at all? For young patients enucleation is probably still mandatory, whereas for older patients enucleation may be optional depending on the patient's overall health. Improved methods of enucleation (no-touch coupled with isolation of the vortex veins, tumor freezing, or hypotensive surgery, or all three) or enucleation combined with preoperative or postoperative radiotherapy or chemotherapy, or both, or newer immunotherapy, may avert the now ominous specter of promoting metastases. Only properly designed and ethical prospective studies will lead us out of our present impasse and clinical uncertainty by assembling the necessary "control groups" of untreated patients and of those managed by modalities other than straight-forward enucleation.

Regardless of the ultimate resolution of the enucleation controversy, we are all indebted to Zimmerman and McLean for sharpening the issues and making us less dependent on conventional wisdom. Not only should the method of enucleation be refined, but a demarche should develop in our thinking and in our research efforts toward a better understanding of the biologic properties of uveal melanomas and the countervailing (or enhancing?) immunologic response to their growth.

FREDERICK A. JAKOBIEC

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AN EVALUATION OF ENUCLEATION IN THE MANAGEMENT OF UVEAL MELANOMAS

Zimmerman and McLean have challenged the value of enucleation in eyes with choroidal melanoma and have presented data suggesting that the procedure may actually disseminate the tumor. Their paper may well prove to be a classic that will change our concepts and management of melanomas of the choroid.

What is the clinician to do until the many questions raised by this exciting presentation are answered? Ideally, one would like to design a study in which randomly selected matched cases would be observed for five, ten, and 15 years while other cases were being enucleated and observed for the same time spans. The extensive education of the public to the necessity of early detection and therapy for cancer precludes such a study even if one were scientifically convinced of its necessity.

I believe, however, that management can be broken down into two general categories. First, that of small tumors (that is, those of less than 10 x 10 x 2mm). This is a subject in which I have long been interested, for at my suggestion, Flocks,[1] reviewed the cases of choroidal melanoma in which histologic specimens were available at the Armed Forces Institute of Pathology. In 1955, he reported that small melanomas had a better prognosis than large melanomas. On the basis of slightly different criteria, this has been confirmed by Jensen[2]; Davidorf and Lang[3]; Shammas and Blodi[4]; and Thomas, Green, and Maumenee.[5] Therefore, because lesions of this size have frequently been found to be either benign or of low-grade malignancy on both histologic and clinical follow-up it seems justifiable to observe these cases after photography and ultrasound examination for definite evidence of growth, especially elevation, before proceeding with invasive therapy. In these cases I am dubious about the necessity or effectiveness of radioactive isotope studies, photocoagulation, or local excision if there is no evidence of growth.

The decsion to treat larger lesions becomes progressively easier as the lesions increase in size, particularly if they have destroyed central vision. Since there is no effective non-invasive medical therapy for malignant melanomas, enucleation of the eye is indicated. Zimmerman and McLean indicate that this should be done as atraumatically as possible. The freezing "no-touch" technique of Fraunfelder and Wilson[6] deserves a well-designed clinical trial. Other techniques also should be sought to reduce massage of the tumor during removal of the eye.

Flaws may possibly be found in some of Zimmerman and McLean's conclusions, but I believe that their evidence for an increase in metastasis and death in the first and second year after enucleation is sufficient to attribute removal of the eye to the increase in mortality. This may occur because of the thin-walled blood vessels or blood lakes in these tumors, which allow malignant cells to be easily pushed into the blood stream by massage. As Zimmerman and McLean have suggested a second possible stimulus for the growth and dissemination of tumor may be an alteration of the immune state. Whatever the cause for the increase in morbidity their study should stimulate many further investigations into this fascinating subject.

A. EDWARD MAUMENEE

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OBITUARY

Jerome T. Pearlman, M.D.
1933-1979

Intelligence, integrity and intrepid good humor were among the qualities that contributed to Jerome T. Pearlman's lifetime of professional distinction and substantial scientific accomplishment. Jerry was internationally known for his research on retinitis pigmentosa, his clinical studies on retinal physiology, and his laboratory investigations of retinal degenerations.

Jerry was born in Chicago on May 11, 1933. He received the Bachelor of Science degree from Dartmouth College and obtained the M.D. degree from Northwestern University in 1957. Thereafter, four years of postdoctoral training in pathology and ophthalmology preceded residency training in ophthalmology at the University of Iowa from 1961 to 1964.

During ophthalmology residency training and for two subsequent years of postdoctoral fellowship at the University of Iowa, Jerry worked under the tutelage of Hermann M. Burian. While engaged in this postresidency training, Jerry received the degree of Master of Science in Ophthalmology. In 1966 and 1967 he completed an additional year of postdoctoral fellowship under the supervision of Frederick Crescitelli at the University of California, Los Angeles. From this fellowship, collaboration with Dr. Crescitelli extended throughout the remainder of Jerry's life.

In 1967, Jerry joined the ophthalmology faculty of UCLA and, at the time of his death Feb. 3, 1979, he held the appointment of professor of ophthalmology and member of the Jules Stein Eye Institute. As a dedicated and extremely effective teacher, he devoted countless hours to the education of medical students, residents, and postdoctoral fellows. As a scholar and investigator, he wrote almost 100 articles on retinitis pigmentosa, retinal electrophysiology, laboratory studies of retinal degenerations, psychosomatic ophthalmology, and the history of ophthalmology. With extraordinary courage and scientific acumen, he pursued his research throughout his illness and, during the final week of his life, completed a major treatise on retinitis pigmentosa.

Honors and distinctions were extended to Jerry throughout his academic career. He was president of the Los Angeles Society of Ophthalmology, vice-president and editor of the International Society for Clinical Electroretinography, recipient of the American Academy of Ophthalmology Honor Award, and an elected member of the Retina Society. In 1978, he was singularly honored by the Southern California Chapter of the Retinitis Pigmentosa Foundation.

These achievements are impressive, but his fine personal attributes loom paramount. He will be remembered with warm affection and deep respect by his many friends and associates.

BRADLEY R. STRAATSMA
FREDERICK C. BLODI

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Substitution of Acetohexamide for Acetazolamide

Editor:

In the article, "Inadvertent substitution of acetohexamide for acetazolamide" (Am. J. Ophthalmol. 84:580, 1977), by N. A. Hargett, R. Ritch, J. Mardirossian, M. A. Kass, and S. M. Podos, the authors describe the serious hazards that can result from this occurrence. Another such incident is detailed below and is offered to underscore the importance of that communication.

CASE REPORT

A 6-day-old male infant was referred for evaluation and treatment of a cloudy left cornea, noted on the day of birth. Bilateral port-wine staining of the skin of the entire face in the typical appearance of the Sturge-Weber syndrome was present. The right eye showed no evidence of glaucoma. Acetazolamide (Diamox), 31 mg every 12 hours, had been given orally as temporary therapy for three days before admission. No untoward effects were noted. Uneventful goniotomy of the left eye was performed on the seventh day of life. Diamox in the same dosage was continued after surgery. A routine screen of blood chemistries included a glucose determination of 64 mg/100 ml on the day of surgery and 82 mg/100 ml on the first postoperative day.

On the fifth day, after completion of hospital discharge procedures, the infant suddenly became lethargic and unresponsive. He responded immediately to a rapid intravenous injection of 50% glucose solution. A blood sample drawn just before treatment indicated a glucose level of 3 mg/100 ml with normal values for sodium, potassium, chloride, carbon dioxide, and urea nitrogen.

We later learned that on the day of his hypoglycemia episode, acetohexamide (Dymelar) had been substituted inadvertently for Diamox by the hospital pharmacy. The nurses in the newborn unit had noted that the medication bottle labelled "Diamox" contained liquid colored differently from an earlier supply.

With appropriate fluid and glucose replacement, including the resumption of acetazolamide, the baby recovered and his condition remained stable.

DISCUSSION

As in Case 2 of Hargett and associates, the pharmacy dispensed acetohexamide in a container labelled "acetazolamide." The substitution was confirmed only retrospectively, after an extreme hypoglycemic reaction which, had it occurred sometime later while the infant and his family were in the midst of their journey home, might have been fatal.

The opportunity for such an occurrence could have been furthered by the unfamiliarity of the nursery staff with either of these drugs, because of its infrequent use in the newborn age group, as well as to their transformation from the familiar tablet form to liquid form. The dose of acetazolamide prescribed, chosen principally to facilitate conversion to a liquid form of the most common tablet size (250 mg), amounted to approximately 18 mg/kg/day, which is higher than the dosage level[1] usually used, although for the treatment of various seizure disorders, doses up to 30 mg/kg/day have been used. This was considered to be a reasonable means of facilitating administration of the agent, in a hospital setting and with the opportunity to monitor blood electrolytes and other biochemical factors. The increase by the same factor of the usual dose level of acetohexamide could have contributed to the severity of the hypoglycemic reaction.

EDWARD L. RAAB, M.D.
New York, New York

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BOOK REVIEWS

Antiviral Agents and Viral Diseases of Man. Edited by George J. Galasso, Thomas C. Merigan, and Robert A. Buchanan. New York, Raven Press, 1979. Hardcover, 732 pages, subject index, preface, 54 tables, 110 black and white figures. $49.50

This book is directed to clinicians as well as to scientists of diverse interests. It will be of special usefulness to medical students and to residents interested in infectious diseases. The book begins with the basic elements of virology including virus structure and replication, the biology of virus infections, the pharmacology of antiviral agents, and the laboratory diagnosis of viral infections. The chapters on the biology of virus infections and on the pharmacology of antiviral agents are particularly informative.

The second part of the book deals with the viral diseases of man and with the treatment of these diseases. The material is divided into discussions of the diseases affecting various organ systems. Among these, Deborah Pavan-Langston's chapter "Ocular Viral Diseases;" is of particular interest. It is both informative and up-to-date. The material is beautifully referenced, and the references are accurate. The diagrams and pictures are highly instructive, although the pictures do not have uniform clarity. I was also somewhat disappointed to find that there was no summary of the clinical trials of various antiviral agents in humans. This would be of special interest for infections such as herpes simplex, herpes zoster, and cytomegalic inclusion disease. Here it would be useful to see a tabular representation of the routes of administration, the dosages of antivirals used, and the data on the effectiveness of each preparation.

On the whole, the book is an excellent one.

G. RICHARD O'CONNOR

Chronic Progressive External Ophthalmoplegia. By L. A. K. Bastiaensen. Winchester, Massachusetts, Sijthoff-Noordhoff International Publishers, 1978. Paperback, 385 pages, table of contents, 134 black and white figures, 15 tables. $24.

Albrecht von Graefe first described chronic progressive external ophthalmoplegia in 1868. This disease is characterized by bilateral blepharoptosis and progressively decreasing ocular motility in all directions of gaze. In the past 20 years investigators of chronic progressive external ophthalmoplegia have associated the ocular muscle defects with deafness, diabetes, ataxia, abnormalities of skeletal muscle, cardiac conduction, and the retinal pigment epithelium.

L. A. K. Bastiaensen, of St. Elisabeth Hospital in Tillburg, the Netherlands, traced all published reports of chronic progressive external ophthalmoplegia from 1868 to 1977. With the addition of several personal contributions in 1978, Dr. Bastiaensen amassed a total of 1,094 cases. Tables detailing the features of these cases occupy half of the book.

Dr. Bastiaensen discusses the anatomy and function of the extraocular muscles, the history and the clinical manifestations of chronic progressive external ophthalmoplegia, and related syndromes. He describes the hereditary, morphologic, and biochemical findings, and presents his own classification of the ophthalmoplegias. In an appendix he includes several of his previous publications on the subject. The reference list is accurate and complete.

This book is the most exhaustive review of chronic progressive external ophthalmoplegia available. It is an important text for anyone studying the syndrome and its diverse manifestations.

ALBERT S. LEVEILLE

Atlas of Eye Surgery and Related Anatomy. Edited by Frederick H. Davidorf. Illustrated by Donald A. Keller. Columbus, Keller Publishing Company, 1978. Hardcover, 289 pages, anatomic index, subject index, 450 black and white figures. $88.

This is a handsome book, beautifully illustrated by Donald A. Keller, medical illustrator of the Department of Ophthalmology of Ohio State University. Additionally, it is published by the Keller Publishing Company and Donald Keller is responsible for the book design and layout. A talented man.

The volume consists of a series of anatomic drawings, followed by a step-by-step illustration of the surgical procedure. A wide variety of techniques are described. A fornix-based flap is used in cataract surgery. After extensive vitreous loss, anterior vitrectomy is done with a cellulose sponge and scissors. Interrupted 10-0 nylon sutures are used for corneal wound closure. The conjunctiva may be closed similarly or with a wet field bipolar cautery. Phacoemulsification, anterior chamber intraocular lenses, and two-loop capsular fixation lenses are described. There is an extensive discussion of postoperative astigmatism including illustrations of knot tying using 10-0 nylon suture. The authors describe cutting of tight suture with a knife to prevent astigmatism. I believe, however, it can be simply done with a laser.

The section on glaucoma leads off with a beautifully illustrated section on gonioscopy. The entire field of ocular surgery is covered with step-by-step illustrations. The Bielschowsky head-tilt test is demonstrated. The combination of the text and illustrations provide both the skilled and the novice surgeons with detailed directions. The trained surgeon may find various steps with which he disagrees, but basic surgical principles are emphasized. The 400 illustrations make the volume particulary valuable.

FRANK W. NEWELL

Duke-Elder's Practice of Refraction, 9th ed. Revised by David Abrams. New York, Churchill Livingstone, 1978. Hardcover, 204 pages, table of contents, preface, appendix, index, 5 tables, 263 black and white figures. $27.50

In the ninth edition of this text, which appears 50 years after the first edition, there is the poignant footnote to the preface, stating that Sir Stewart died during the final stages of the preparation. It is fortunate for later generations of ophthalmologists that Mr. David Abrams who collaborated with Sir Stewart so effectively, is available to carry on.

The first half of this edition, dealing with optics and physiologic optics, refractive errors, accommodation, and binocular vision and its anomalies is relatively unchanged from the eighth edition except for some logical changes in the order in which they are discussed.

The second half has been generally rewritten. It covers clinical investigation including retinoscopy and subjective verification and optical appliances. Unfortunately the explanation and particularly the illustrations of the optics of retinoscopy are confusing. It is remarkable that the optics of a technique so commonly used should be so difficult to explain.

Mr. Abrams is eloquent in his advocacy of noncycloplegic retinoscopy, a procedure I find particularly useful. It is my impression that most ophthalmologists in the United States use a cycloplegic routinely before retinoscopy. Mr. Abrams believes the use of such drugs is "not an unmixed blessing."

The one great advantage of streak retinoscopy, which is now used so widely in the United States either with or without a cycloplegic, is the definition of both the strength and the axis of any cylinder that may be present. The behavior of the retinoscopic reflex when the correcting cylinder is at the incorrect axis is not described in detail.

In the subjective verification of retinoscopic findings, the crossed cylinder is invaluable in determining both axis and strength. Mr. Abrams' explanation of this procedure will be disappointing to those who learned refraction from the late Jack Copeland. Mr. Copeland taught that the power and axis of the cylinder was determined by retinoscopy and that this information was "refined" by the use of the crossed cylinder. In refining the power of the correcting cylinder, he maintained that the eye seized on the circle of least diffusion of the conoid of Sturm. It was his contention that the proper technique is to collapse any remaining conoid onto the retina by allowing the anterior focal line to go back toward the retina with a concave sphere, and bringing the posterior line forward toward the retina by using a convex cylinder of twice the power. Mr. Abrams does not mention this procedure.

The section on best form lenses is interesting but it is unfortunate that new material on the best form of aphakic lenses is not included.

It is easy for a reviewer to single out passages with which he disagrees, but this should not detract for this is a splendid book full of information for the beginning refractionist. It is highly recommended and I hope that Mr. Abrams will continue to bring out new editions as needed for the next 50 years.

DANIEL SNYDACKER

Bernard Becker, M.D., Collection in Ophthalmology. Cataloging by Mark Weimer. Editing and Cataloging Introduction by Christopher Houlihan. Washington University School of Medicine, St. Louis, 1979. Paperbound, 102 pages, geographical index, index of printers & publishers, Microfiche of title pages, $12.50

In 1975, Bernard Becker, Professor and Chairman of the Department of Ophthalmology at Washington University Medical School, presented his collection of ophthalmic rarities to the University Library. The university in turn constructed a new rare book annex and provided special facilities for the housing of the ophthalmic collection. Since then Becker has continued to add to the collection and also advised the library on the purchase of new material.

Mr. Mark Weimer catalogued the Becker collection from 1975 to 1977 and this has been carried on since then by his successor, Mr. Christopher Houlihan. In addition to the catalog, a photographic copy of the title of each work has been made into a Microfiche and included with the volume.

The catalog contains some 268 items ranging from Abu Bakr al-Hasan ibn al-Khasib, through George James Guthrie to Johann Gottfried Zinn. Additionally, 13 prints are described and a catalog on duplicates of less rare items is provided. The oldest item is from 1496, Petrus Lacepiera, which is an Italian translation of the second earliest printed work on the eye. The most recent is Pierre Armand Dufau in 1850, being an important contribution to the theory and practicing of educating the blind to read and learn useful manual skills.

All in all it was unusually well done and we are indebted to Dr. Becker for bringing the collection together, and to Washington University for providing such a handsome record.

FRANK W. NEWELL

SYMPOSIA

Third International Symposium on Orbital Disorders. Edited by G. M. Bleeker. The Hague, Dr. W. Junk bv Publishers, 1978. Hardcover, 546 pages, table of contents, subject index, 84 tables, 311 black and white figures, 2 color figures. $87.50

J. FRANÇOIS AND F. GOES *(Belgium):* **A-scan ultrasonography and diagnosis of unilateral exophthalmos**

K. C. OSSOINIG *(USA):* **The role of clinical echography in modern diagnosis of orbital and periorbital lesions (abstract)**

M. RESTORI, J. E. WRIGHT, AND D. MCLEOD *(England):* **B-scan and C-scan imaging in the orbit**

H. W. BUSCHMAN AND D. LINNERT *(Fed. Rep. Germany):* **Ultrasound exophthalmometry, orbital echography and CAT in diagnosis of orbital disorders**

A. KANEKO *(Japan):* **B-scan ultrasonography of rhinogenic exophthalmos**

J. POUJOL AND N. TOUFIC *(France):* **Combined A- and contact B-scan ultrasonography of orbital disease**

W. SCHROEDER *(Fed. Rep. Germany):* **Ultrasonography of the optic nerve**

P. A. GOMMERS, G. BLAAUW, AND R. WIJNGAARDE *(The Netherlands):* **Unilateral high myopia: comparison of ultrasonography and CT-scanning**

E. A. CANABIS, M. T. IBA-ZIZEN, V. DANICEL, AND A. LOPEZ *(France):* **Radiological diagnosis of orbital fractures in ophthalmology**

C. CLAY, P. LASJAUNIAS, J. MORET, AND J. VIGNAUD *(France):* **Theoretical ophthalmologic risk of embolization**

F. PEETERS, R. KROGER, B. VERBEETEN, JR., AND C. VERSTEEGE *(The Netherlands):* **The value of computer tomography (CT-scanning), orbital venography and carotid angiography in the diagnosis of exophthalmos**

P. LASJAUNIAS AND J. MORET *(France):* **Varying origin of the ophthalmic artery**

C. DERREMEAUX, C. CLAY, P. LASJAUNIAS, AND J. VIGNAUD *(France):* **Radiographical anatomy of the musculosensorial vessels (abstract)**

M. T. IBA-ZIZEN, E. A. CABANIS, P. BONNIN, AND G. PORRET *(France):* **Dynamic angiography of the orbit**

S. L. TROKEL AND S. K. HILAL *(USA):* **Thin section computerized tomography: analysis of 600 orbit studies**

G. A. S. LLOYD AND J. AMBROSE *(England):* **A** comparative evaluation of computerized tomography in orbital diagnosis

J. VIGNAUD AND M. L. AUBIN *(France):* Coronal sections in C.T. of the orbit

A. S. GROVE, JR., R. TADMOR, K. J. MOMOSE, AND P. F. J. NEW *(USA):* Computerized tomography for evaluation of fractures and foreign bodies of the orbit

F. BIGAR, H. SPIESS, AND C. BOSSHARD *(Switzerland):* Standardized A-scan echography and computerized tomography for evaluation of orbital disease

A. NOVER AND J. SCHMITT *(Fed. Rep. Germany):* Misleading findings obtained by ultrasonography and computerized tomography

S. LANGE, TH. GRUMME, S. WENDE, K. KRETZSCHMER, E. KAZNER, AND H. STEINHOFF *(Fed Rep. Germany):* Computerized tomography in orbital diseases

U. SALVOLINI, A. RODALLEC, E. A. CABANIS, F. MENICHELLI, U. PASQUINI, AND P. BONNIN *(Italy and France):* C.T. of the optic nerve in papilledema

A. ROUSSEAU, J. MICHIELS, G. CORNELIS, AND H. H. VANDRESSE *(Belgium):* Computed tomography in ophthalmology

G. BLAAUW, W. H. BAKKER, P. P. M. KOOY, AND R. WIJNGAARDE *(The Netherlands):* Technetium scanning in orbital disorders

M. PASSOT, P. BONNIN, M. T. IBA-ZIZEN, AND E. A. CABANIS *(France):* Plate thermography: angiographic correlation in orbital and cephalic disorders

G. BONAVOLONTA, G. CENNAMO, P. ROCCO, F. MENICHELLI, U. PASQUINI, AND U. SALVOLINI *(Italy):* C.T. echography and thermography in the diagnosis of unilateral exophthalmos

H. A. SISLER *(USA):* Instrumental visual axis ophthalmodynamometry

C. T. BUITER *(The Netherlands):* Peri-orbital endoscopy

A. B. DE HAAN *(The Netherlands):* An accessory muscle in the left orbit of a foetus of 123-mm crown-rump length

M. P. BERGEN AND J. A. LOS *(The Netherlands):* Vascular patterns in the human orbit in relation to the connective tissue septa

CHR. VERMEIJ-KEERS *(The Netherlands):* The orbital muscle and the cavernous sinus

L. KOORNNEEF *(The Netherlands):* The connective tissue apparatus of the human orbit. What about it?

G. OUDHOF *(The Netherlands):* Quantitative approach to the estimation of the relationship of the growth patterns of eye and orbit

J. BRIHAYE, M. VAN GEERTRUYDEN, R. HERZEEL, AND B. WILLEKENS *(Belgium and The Netherlands):* Cysts of the optic nerve sheets

D. LEBUISSON, P. DICONSTANZO, J. J. ARON-ROSA *(France):* Optic nerve hypoplasia (abstract)

B. D. DE JONG AND K. E. W. P. TAN *(The Netherlands):* Facial clefts in combination with anophthalmus on tear duct anomalies. The possibilities of plastic reconstruction

B. D. DE JONG *(The Netherlands):* Eyelid reconstruction in facial clefts (abstract)

H. MUHLENDYCK AND D. LEITHAUSER *(Fed. Rep. Germany):* Diagnostic problems in cases with blow-out fractures and motility disturbances of other origin

T. FUJINO AND T. B. SATO *(Japan):* Mechanisms, tolerance limit curve and theoretical analysis in blowout fractures of two and three-dimensional orbital wall models

G. J. KUSEN, H. P. VAN DEN AKKER, AND M. BAZUIN *(The Netherlands):* Stable fixation of fractures of the zygomatic bone with the use of AO compression plates (abstract)

F. HOLLWICH, H. BUSSE, AND H. P. SCHIFFER *(Fed. Rep. Germany):* The blow-out fracture in the view of an ophthalmologist

Y. FUKADO *(Japan):* Surgical treatment of blowout fracture

Y. FUKADO *(Japan):* Surgical decompression of blowout fracture (abstract of film)

A. MORTADA *(Egypt):* Proptosis due to parasitic infestations

I. LEVY *(England):* Acute infections of the orbit

E. WEIGELIN AND U. KLEHR *(Fed. Rep. Germany):* Mucormycosis of the orbit

G. IRACI, L. TOMAZZOLI GEROSA, AND M. GEROSA *(Italy):* Optochiasmatic arachnoiditis combined with an intra-orbital inflammatory mass lesion (Case report)

A. HUBER *(Switzerland):* Metastatic orbital abscess, an unusual observation

R. M. CHAVIS, A. GARNER, AND J. WRIGHT *(USA and England):* Inflammatory orbital pseudotumor—a clinical pathological study

R. K. TAMIN RADJAMIN *(Indonesia):* Eye tumors in Dr. Soetomo Hospital Surabaya, Indonesia

B. SCHMIDT *(Fed. Rep. Germany):* Occurrence and frequency of eye tumors in Ethiopia

H. CHAMS AND A. KHEYRIEH *(Iran):* Six hundred and eighty-four tumors of the eye and the orbit treated in the Farabi Eye Hospital over the last five years

E. A. VAN SLOOTEN *(The Netherlands):* Lymphoreticular disease with involvement of the orbital wall? The value of selective bone biopsy

J. P. A. GILLISSEN AND H. J. F. PEETERS *(The Netherlands):* Intra-orbital tumors of the peripheral nerves

H. J. F. PEETERS AND J. P. A. GILLISSEN *(The Netherlands):* Benign tumors of the bony orbital wall

H. MATUSO AND M. USUI *(Japan):* Five cases of nasal sinus mucoceles and their ocular styptoms

D. COSTER AND J. E. WRIGHT *(England):* The orbital manifestations of von Recklinghausen's disease

F. A. JAKOBIEC, G. D. POTTER, J. MITCHELL, AND I. S. JONES *(USA):* Ossifying fibromas of the orbital roof

O. FISCHEDICK AND K. ULLERICH *(Fed. Rep. Germany):* In vitro and in vivo diagnosis of endocrine ophthalmopathy

N. F. LAWTON, P. FELLS, AND G. A. S. LLOYD *(England):* Medical investigation of dysthyroid eye disease

W. M. WIERSINGA AND J. L. TOUBER *(The Netherlands):* The relevance of the TRH-test in the differential diagnosis of exophthalmos

Y. INOUE AND T. INOUE *(Japan):* Study on dysthyroid ophthalmopathy

H. KRUSE AND W. RUSSMANN *(Fed. Rep. Germany):* Management of vertical squint in endocrine orbitopathy

R. J. W. DE KEIZER *(The Netherlands):* A case report on a 15-year-old girl with hyperthyroidism and myasthenia gravis. Discussion of diagnosis and etiology on the relation between these diseases

J. BRISMAR AND G. BRISMAR *(Sweden):* Spontaneous carotid-cavernous fistulas and aseptic cavernous sinus thrombosis—two related disorders

P. DWYER JONES, P. EUSTACE, AND J. TOLAND *(Ireland):* The Tolosa Hunt syndrome

D. ARON-ROSA AND D. DOYON *(France):* The Tolosa Hunt syndrome

G. BRISMAR AND J. BRISMAR *(Sweden):* Aspetic venous thrombosis or Tolosa Hunt syndrome: discussion of diagnostic criteria with reference to 12 personal cases

T. T. HUANG AND S. R. LEWIS *(USA):* Blindness associated with retrobulbar hemorrhage

F. OSMERS, H. BUSSE, AND H. P. SCHIFFER *(Fed. Rep. Germany):* Aspects on the diagnosis of venous malformation of the orbit

J. T. W. VAN DALEN *(The Netherlands):* The Tolosa Hunt syndrome (painful ophthalmolplegia) (abstract)

B. SMITH *(USA):* Herniated lacrimal glands and the technique of suspension

I. SNOW JONES *(USA):* Lateral orbital decompression

A. CASTERMANS, G. LAVERGNE, AND A. VAN GARRSE-LYSENS *(Belgium):* The surgical treatment of endocrine exophthalmos

V. H. SMITH *(England):* Experiences with medial decompression of the orbit

M. A. H. EL ASWAD *(Egypt):* Lacrimal protheses (El Aswad prothesis)

J. C. H. VAN DER MEULEN *(The Netherlands):* Orbital recession in cranofacial surgery (abstract)

T. H. OEI *(The Netherlands):* Surgical approach to the orbital connective tissue in cases of traumatic diplopia

K. MULLER-JENSEN *(Fed. Rep. Germany):* Foreign body reaction after orbital Teflon implatation

D. MARCHAC, J. COPHIGNON, J.-F. HIRSCH, AND D. RENIER *(France):* Correcting the deficiency of the supra orbital rim in craniostenosis by radical osteotomies, about 28 cases (abstract of film)

J. WRIGHT *(England):* The surgical management of orbital tumors (abstract)

A. CALLAHAN *(USA):* Mucocoele of maxillary sinus (abstract of film)

P. A. VOUTE AND J. DE KRAKER *(The Netherlands):* **Medical treatment of malignant orbital tumors in children**

J. SCHIPPER AND K. E. W. P. TAN *(The Netherlands):* **An accurate and simple technique for megavoltage therapy of intra-orbital tumors**

P. LOMMATZSCH AND S. MAU *(German Dem. Rep.):* **Radiation therapy of orbital tumors**

J. T. W. VAN DALEN, A. M. FRENS, AND G. P. M. HORSTEN *(The Netherlands):* **The visual evoked response as a simple diagnostic procedure in cases of optic nerve and chiasmal lesions**

R. WIJNGAARDE AND G. BLAAUW *(The Netherlands):* **Folds in the posterior pole of the eye in orbital disorders**

J. D. BULLOCK AND R. R. WALLER *(USA):* **Choroidal folding in orbital disease**

J. P. WITMER *(The Netherlands):* **An ERG indication of the existence of optic nerve lesions**

J. L. VROOLAND *(The Netherlands):* **Isometric contraction of the extraocular muscles**

KARL C. OSSOINIG *(USA):* **The role of clinical echography in modern diagnosis of orbital and periorbital lesions**

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FRANK W. NEWELL, M.D.
Editor-in-Chief
AMERICAN JOURNAL OF OPHTHALMOLOGY

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

American Journal of Surgery

SCREENING FOR SIGNIFICANT CAROTID STENOSIS BY OCULAR PNEUMOPLETHYSMOGRAPHY. McDonald, K. M., Gee, W., Kaupp, H. A., and Bast, R. G. (Dept. Surg., Allentown and Sacred Heart Hosp. Ctr., Allentown, Pa.). Am. J. Surg. 137:244, 1979.

Accurate measurement of the systolic pressure in the distal segments of the arterial system has become an accepted method of assessing the functional significance of proximal atherosclerotic stenosis. The ophthalmic artery is a distal segment of the internal carotid artery, but the circle of Willis provides collateral channels unique to the respective ophthalmic arteries. Therefore, measurement of the systolic pressure in both ophthalmic arteries simultaneously is essential. An instrument which will do this is the ocular pneumoplethysmograph and this report describes its use to measure both ophthalmic systolic pressures simultaneously and correlates these data with systemic systolic pressures measured by arm cuff and auscultation. If one assumes that carotid stenosis is significant when the resulting lumen is 50% or less of the diameter of the vessel distal to the carotid sinus than the ocular pneumoplethysmograph can detect these stenoses with an accuracy of 97%. (5 figures, 2 tables, 14 references)—David Shoch

Annals of Surgery

OCULAR PNEUMOPLETHYSMOGRAPHY: DETECTION OF CAROTID OCCLUSIVE DISEASE. McDonald, P. T., Rich, N. M., Collins, G. J., Kozloff, L., and Andersen, C. A. (Dept. Surg., Walter Reed Army Med. Ctr., Washington, D.C.). Ann. Surg. 189:44, 1979.

To determine the accuracy of ocular pneumoplethysmography in detecting carotid arterial occlusive disease, 350 patients were tested. Sixty-three patients underwent angiography and the findings were correlated with the results of ocular pneumoplethysmography. Testing without carotid compression averaged three minutes and was easily performed by a physician or technician. There were no significant complications. Hypertension did not affect evaluation. There were two false-negative tests and no false-positive tests. Without carotid compression the overall accuracy for testing for significant arterial stenosis was 97%. When a carotid compression test was added, the two missed lesions were detected. (5 figures, 1 table, 7 references)—Authors' abstract

Brain

HEMISPHERE FUNCTION AND BINOCULAR DEPTH PERCEPTION. Danta, G., Hilton, R. C., and O'Boyle, D. J. (Neurol. Dept., Manchester Royal Infirm., and Dept. Psychol., Univ. Manchester, England). Brain 101:569, 1978.

Binocular depth perception was measured in 26 patients with right and 28 patients with left hemisphere lesions. In general among clinical signs only the presence of visual field defects correlated with abnormalities of the visual depth perception. One patient, however, who had a complete left homonymous hemianopia with macular sparing, had normal stereoacuity. Lesions clinically not implicating the primary visual pathways and situated remote from the occipital lobes often impaired visual depth perception. The authors therefore conclude that: 1) disorders of stereopsis and visual depth perception can be demonstrated in a large proportion of patients with focal cerebral hemispheric lesions, but only uncommonly does a hemispheric lesion give rise to a symptomatic and significantly disabling disorder of depth perception; 2) although visual field defects are common in patients with hemispheric lesions and disordered depth perception, there is no evidence that any particular neural or ocular signs are necessarily of a etiological significance in the production of the disorder; 3) neural processes underlying binocular single vision (fusion) and binocular depth discrimination may be functionally dissociable; 4) neural mechanisms in areas outside the occipital lobes and primary visual projection systems seem to play an important role in normal stereopsis; visual depth perception seems not to depend on the integrity of a unitary brain mechanism discretely localized within a particular area of one hemisphere, but rather upon the hierarchical organization of a number of logically discrete but functionally interrelated neural mechanisms which may involve widespread areas of the brain; 5) the question of whether or not depth discrimination and stereopsis is a function to which hemisphere dominance applies is as yet unsettled. On available evidence it cannot be unequivocally stated that the right hemisphere is dominant for stereopsis; 6) interpretation of systematic studies of stereoacuity and visual depth perception is made difficult by the fact that results derived from different tests are often not comparable. (4 figures, 5 tables, 59 references)—Authors' abstract

MEASUREMENTS OF VISUAL EVOKED POTENTIALS IN PARKINSON'S DISEASE. Bodis-Wollner, I., and Yahr, M. D. (Dept. Neurol., Mount Sinai School of Med., New York, N.Y.). Brain 101:661, 1978.

Visual evoked potentials elicited by reversing grating patterns were recorded in 35 patients with Parkinson's disease and in 26 controls. The average latency of evoked potentials in patients with Parkinson's disease exceeded by two standard deviations the average of the age-matched control group of other neurological patients. Over two-thirds of all patients with Parkinson's disease had abnormal latency. In these patients latency did not correlate with age. In five patients the latency became less prolonged on levodopa therapy, suggesting that catecholaminergic pathways have either indirect or direct effects on the generation of visual evoked potentials. (6 figures, 19 references)—Authors' abstract

Brain Research

MONOCULAR AND BINOCULAR DEPRIVATION IN THE MONKEY: MORPHOLOGICAL EFFECTS AND REVERSIBILITY. Vital-Durand, F., Garey, L. J., and Blakemore, C. (Inst. Anat., Univ. of Lausanne, Switzerland). Brain Res. 158:45, 1978.

Measurements were made of the cross-sectional area of neurons in the lateral geniculate nucleus (LGN) of normal monkeys, and of monkeys subjected to monocular and binocular eyelid suture. Early monocular deprivation caused a failure of normal cell growth in the LGN such that the neurons in the laminae innervated by the deprived eye were, on average, 15% smaller than those innervated by the normal eye. In the first four days of life monocular deprivation caused significant retardation of growth. The effect was marked in the first six weeks, but was not found in a monkey monocularly deprived from 11-16 months of age nor in an adult deprived for more than six months. Binocular deprivation from birth appeared to arrest neuronal growth at the neonatal size.

The effect of monocular deprivation could be cancelled by 'reverse-suture' (opening the closed eye and closing the other) during the first two months of life.

Changes in the size of LGN neurons following monocular deprivation and reverse-suture correlated closely with changes in the relative width of ocular dominance columns in layer IVc of area 17 of the visual cortex, measured physiologically in the same animals. (10 figures, 1 table, 38 references)—Authors' abstract

British Journal of Ophthalmology

PATHOGENESIS AND CLASSIFICATION OF MASSIVE PERIRETINAL PROLIFERATION. Machemer, R. (Bascom Palmer Eye Inst., Dept. Ophthalmol., Univ. of Miami, School of Med., and Veterans Admin. Hosp., Miami, Florida). Br. J. Ophthalmol. 62:737, 1978.

A unifying concept of massive periretinal proliferation (MPP) has been developed from three sources of information: the experimentally produced retinal detachment in the owl monkey, the analysis of intraocular situations during vitreous surgery and pathological studies of human eyes. The histological processes leading to MPP are: 1) proliferation of the cells of the bared pigment epithelium with concurrent transformation into wandering macrophages and fibroblast-like cells which form cluster or sheets on any surface they can find, 2) proliferation of retinal glial cells which grow unto the inner or outer retinal surface where they form membranes, and 3) contraction of such membranes the mechanism of which is similar to the fibroblastic contraction occurring in wound repair in other tissues. In the evolution of MPP four stages can usually be recognized of which the first two are diagnostically most important. Stage I is characterized by diffuse vitreous haze with a marked increase in the number of fine brown dots in it; these are isolated pigment epithelial macrophages which may also be seen on everted flaps of retinal breaks and later on the anterior surface of the inferior retina. In stage II the brown dots turn into less pigmented clusters or clumps which are the first signs of beginning "stickiness" of the proliferating cells. In stage III cellular membranes make their appearance on all available surfaces including the posterior surface of the detached vitreous. Stage IV is that of contraction of the membranes and consecutive rigid folding of the retina. The location of the membrane, preretinal or retroretinal, determines the configuration of the retinal folds. The disease may come to a halt at stage I or become explosive, creating within hours the full-blown picture of MPP. (17 figures, 28 references)—Peter C. Kronfeld

THE O'CONNOR CINCH REVISITED. Williams, A. T., Metz, H. S., and Jampolsky, A. (Smith-Kettlewell Inst. of Visual Science, San Francisco, Calif.). Br. J. Ophthalmol. 62:765, 1978.

The authors briefly review the history of the cinch operation, describe their technique and present results in normal dogs and in 17 human cases in whom cinching of one horizontal rectus, usually combined with weakening of the antagonist by recession of "Z" myotomy, was used as part of the surgical procedure. The average preoperative deviation in that group was 28 PD with a range from 80 PD of exotropia to 40 PD of esotropia. Adjustments, i.e., removal of one or more of the eight strands of nylon used as core of the cinch, were made on the basis of cover tests in four cases within 24 hours and in two cases six days postoperatively. The average residual deviation after a mean postoperative period of three and a half months was two PD, with a range from ten PD exotropia to 12 PD of esotropia. (4 figures, 5 references)—Peter C. Kronfeld

MORPHOLOGICAL CHANGES IN THE HUMAN CONJUNCTIVAL EPITHELIUM. I. IN THE NORMAL ELDERLY POPULATION. II. IN KERATOCONJUNCTIVITIS SICCA. Abdel-Khalek, L. M. R., Williamson, J., and Lee, W. R. (Depts. Ophthalmol. and Pathol., Univ. of Glasgow, and the Southern General Hosp., Glasgow). Br. J. Ophthalmol. 62:792 and 800, 1978.

Biopsies of the bulbar conjunctiva about five millimeters from the upper limbus were taken in connection with routine cataract surgery. In conjunctivas free of clinical disease light microscopy confirmed the classical description of a three-layered epithelium with a fairly constant number of goblet cells in the superficial layer; individuals over 80 years of age showed a significant reduction in the number of goblet cells and, in four out of 16 specimens, abnormal, round or ovoid acellular structures which were considerably larger than the goblet cells and stained positively with the PAS stain (named "hyaline bodies" by the authors). A new electron microscopic finding was the presence of rod-shaped cytoplasmic organelles in the intermediate epithelial layer closely resembling structures commonly seen in the endothelial cells of the vascular system. The principal findings in 24 patients suffering from keratoconjunctivitis sicca were stratification of the superficial epithelial layer with widening of the intercellular spaces and almost complete disappearance of the goblet cells. The magnitude of these changes was directly proportional to the clinical severity of the disease. The histological manifestations could be reversed by surgical occlusion of the lower lacrimal canaliculus. (11 figures, 3 tables, 23 references)—Peter C. Kronfeld

ACUTE FOLLICULAR CONJUNCTIVITIS AND KERATOCONJUNCTIVITIS DUE TO HERPES SIMPLEX VIRUS IN LONDON. Darougar, S., Hunter, P. A., Viswalingam, M., Gibson, J. A., and Jones, B. R. (Dept. Clin. Ophthalmol., Inst. of Ophthalmol. and External Disease Clin., Moorfields Eye Hosp., London). Br. J. Ophthalmol. 62:843, 1978.

Twenty-five patients presented with an acute follicular conjunctivitis resembling adenovirus infection. In five cases there had been antecedents in the form of mild pharyngitis and rhinitis. The ocular disease was characterized by mild discharge, mild to moderate hyperemia, moderate to severe follicular hypertrophy in the fornices and over the tarsi and severe papillary hypertrophy over the tarsi. The cornea was involved in two forms: coarse epithelial punctate keratitis, which developed early in nine cases, and subepithelial infiltrates resembling those of epidemic keratoconjunctivitis and persisting up to four months in five cases. Clinical signs typical for herpes simplex (vesicles or ulcers on the lid borders or small dendritic corneal ulcers) were noted in five cases. In the remaining 20 cases the correct etiological diagnosis was made only by virus culture (apparently on human embryonic kidney cells) or by a four-fold or greater rise in the level of herpes simplex antibody in the serum. Antiherpetic chemotherapy seemed to shorten the course of the disease. (9 figures, 1 table, 12 references)—Peter C. Kronfeld

Chinese Journal of Ophthalmology

EXPERIENCE IN 2002 CASES OF INTRAOCULAR FOREIGN BODY EXTRACTION. Chang, H., Sung, H. W., Yang, C. T., Cheng, L. N., Shih, P., and Chi, L. S. (Honan Med. School, First Hosp.). Chinese J. Ophthalmol. 14:11, 1978.

During the past 22 years, 2014 human eyes were observed after removal of intraocular foreign bodies. Of the subjects, 92.4% were male and 7.6% were female, the age range being two to 63 years. Approximately 80% of the foreign bodies were magnetic and the remainder nonmagnetic. Patients were generally industrial workers (61.42%) or farmers (26.30%). The two most frequent circumstances of injury were hammering (79.07%) and explosion (15.32%). Left eyes were injured more often than right eyes. Foreign bodies were in the posterior segment of the eye in 59.2% of the cases, in the anterior chamber in 29.2%, and on the eye wall in 11.6%. The foreign bodies were removed through the posterior segment or by pars plana incision in 85% of the cases and by an incision of the cornea or the limbus in 15%. Retinal detachment was seen in 63 eyes before surgery and in 39 after surgery. (2 references)—Luke H. Liu

CLINICAL OBSERVATIONS ON 712 CASES (839 EYES) OF LASER IRIDECTOMY. (Dept. Ophthalmol., Second People's Hosp., Jinan, Shandung). Chinese J. Ophthalmol. 14:25, 1978.

A Chinese-made YJH-1-type ruby laser was used to perform an iridectomy in 712 cases, involving 839 eyes, over the past two years. Three hundred and eighteen patients were male and 394 female. Age distribution was four to 78 years. Operations were for congestive glaucoma in 461 eyes and for optical iridectomy in 255. Most patients were observed at six months, and some were checked two years after the procedure. After operation on the 461 congestive-glaucoma eyes, intraocular pressure returned to normal or near-normal in 397 eyes (86.1%)—without need of additional medication in 288 eyes and with additional local application of antiglaucoma drops in 109. After optical iridectomy in 255 eyes, vision was improved in 189 (74.1%). The following complications were seen during or after laser iridectomy: burn of the corneal epithelium (58 eyes), damage of the corneal endothelium (34 eyes), hyphema (25 eyes), fibrinous exudates around iridectomized area of iris (23 eyes), mild opacities on anterior capsule of lens (8 eyes), and injury of retina (1 eye). (1 figure, 2 tables, 5 references)—Luke H. Liu

Journal of the American Medical Association

MICROSURGICAL TREATMENT OF TRANSIENT CEREBRAL ISCHEMIA. Samson, D. S., Hodosh, R. M., and Clark, W. K. (Div. Neurosurg., Univ. Texas Health Sci. Ctr. Hosp., Dallas, Tex.). J.A.M.A. 241:376, 1979.

Extracranial-intracranial arterial bypass is a microneurosurgical procedure recently introduced in the treatment of a variety of cerebrovascular ischemic states. Fifty patients with transient ischemic attacks (TIAs) localized to the distribution of the internal carotid artery underwent this procedure during a 48-month period. All have been followed up for at least 14 months after surgery. There were no operative deaths, and notable postoperative morbidity has been experienced in less than 8% of cases. Seventy-six percent of patients have been asymptomatic since surgery, 14% have continued to experience TIAs, and 6% have had completed strokes (2% occurring in the operative hemisphere). (1 figure, 1 table, 20 references)—Authors' abstract

OCULAR FINDINGS IN BOTULISM TYPE B. Terranova, W., Palumbo, J. N., and Breman, J. G. (Ctr. for Disease Control, Atlanta, Georgia and Dept. Ophthalmol., Mount Sinai School of Med., New York, N.Y.) J.A.M.A. 241:475, 1979.

An outbreak of type B botulism occurred in a cohort of 59 persons all of whom had eaten in the same restaurant. All 59 complained of dry mouth and 51 of the 59 (86%) noted difficulty in focusing at near or complained of diplopia. Forty-five of the patients developed neurologic findings and in 44 of these 45 there were bilateral cranial sixth nerve palsies. The fourth nerve was involved only in 18% and the third nerve in 22%. The signs of third nerve palsy were ptosis, dilated sluggish pupils and medial rectus paralysis. It is of interest and clinical import that eight of the 11 persons who had all three signs of third nerve involvement developed respiratory insufficiency. One patient who did not have all three findings also developed respiratory difficulty and this was out of 34 patients. Therefore, the presence of severe oculomotor nerve paralysis is a diagnostic and prognostic factor for the development of respiratory insufficiency and should alert one to this possibility. (1 table, 8 references)—David Shoch

Neurology

A PROSPECTIVE STUDY OF THE RISK OF DEVELOPING MULTIPLE SCLEROSIS IN UNCOMPLICATED OPTIC NEURITIS. Cohen, M. M., Lessell, S., and Wolf, P. A. (Depts. Ophthalmol., Neurol., and Anat., Boston Univ. School of Med., Boston, Mass.). Neurology 29:208, 1979.

• Sixty patients with uncomplicated optic neuritis were followed for at least five years to determine the risk of developing subsequent multiple sclerosis. The average follow-up time was 7.1 years. In this period 17 patients (28%) developed definite multiple sclerosis and four (7%) developed probable or possible multiple sclerosis. Six of the 17 patients who developed definite multiple sclerosis did so in the first year. The incidence appeared to be greater in women in that 45% of the women but only 11% of the men developed multiple sclerosis. Both sexes were at highest risk if the optic neuritis occurred between the ages of 21 and 40. Fifty-one percent of the patients in this age group progressed to multiple sclerosis, whereas the risk in other age groups was 12%. There was an overall increased risk of multiple sclerosis with recurrent optic neuritis. The course of the multiple sclerosis itself appeared to be benign during the period of observation. (2 figures, 2 tables, 39 references)—David Shoch

INTERNUCLEAR OPHTHALMOPLEGIA CAUSED BY SUBDURAL HEMATOMA. Deveraux, M. W., Brust, J. C. M., and Keane, J. R. (Dept. Neurol., Harlem Hosp. Ctr., New York, N.Y.). Neurology 29:251, 1979.

Internuclear ophthalmoplegia is caused by lesions in the median longitudinal fasciculus. It is generally the result of primary intraaxial disorders, most commonly multiple sclerosis in young adults and infarction in older adults. Rarely, extraaxial disorders cause internuclear ophthalmoplegia by compressing the brainstem. The authors report two patients with internuclear ophthalmoplegia resulting from subdural hematoma with transtentorial herniation. These cases demonstrate that internuclear ophthalmoplegia is a reliable, but not a pathognomonic, sign of a primary intraaxial disorder. (5 figures, 21 references)—Authors' abstract

New England Journal of Medicine

CARDIAC EFFECTS OF TOPICAL TIMOLOL. Britman, N. A. (Plainfield, N.J.). N. Engl. J. Med. 300:566, 1979.

A 72-year-old woman with severe rheumatic heart disease was marginally compensated on digoxin and diuretics. She was placed on timolol, 0.25% b.i.d. and within two weeks was in severe congestive heart failure. Initially she was felt to have digoxin toxicity and this drug was stopped with no improvement. A week later the possibility that timolol was causing the problem was brought up and the drug was stopped. In three days the patient lost 3.7 kg and her ventricular rate increased to normal. The presence of heart disease should be questioned and noted in any patient receiving topical beta-adrenergic receptor blockers.—David Shoch

HUMAN-TO-HUMAN TRANSMISSION OF RABIES VIRUS BY CORNEAL TRANSPLANT. Houff, S. A., Burton, R. C., Wilson, R. W., Henson, T. E., London, W. T., Baer, G. M., Anderson, L. J., Winkler, W. G., Madden, D. L., and Sever, J. L. (Infectious Diseases Branch, Natl. Inst. Neurol. and Communicative Disorders and Stroke, Natl.

Inst. Health, Bethesda, Md.). N. Engl. J. Med. 300:603, 1979.

A 39-year-old man died of a undiagnosed generalized neurologic disorder. The cornea of one eye was then used for transplantation into the eye of a 37-year-old woman. About a month after the transplantation, neurologic symptoms developed in the patient, similar to the symptomatology of the donor. The patient gradually became comatose and died 50 days after the corneal transplantation. Pathologic and virologic studies were made not only of the tissues from the woman but from the frozen eye that had been retained from the donor. In all tissues rabies virus was demonstrable by immunofluorescence and mice that were inoculated with tissue taken from both the donor and the recipient developed rabies. Several years ago the probable transmission of Creutzfeldt-Jakob disease was reported and now rabies virus appears to be transmissible via corneal transplantation. Tissues from patients with neurologic disease suspected of having either a viral or "immune" pathogenesis should not be used for transplantation. (17 references)—David Shoch

Science

RETINOBLASTOMA WITH 13q- CHROMOSOMAL DELETION ASSOCIATED WITH MATERNAL PARACENTRIC INVERSION OF 13q. Sparkes, R. S., Muller, H., Klisak, I., and Abram, J. A. (Div. Med. Genetics, and Depts. Med., Pediatr., and Psychiatry, Ctr. Health Sci., Univ. California, Los Angeles, Calif.). Science 203:1027, 1979.

Most retinoblastomas occur sporadically while a small number are inherited in a dominant fashion. A third small group has been found to have a partial deletion of the long arm of chromosome 13 (13q-). Such a patient is reported here but it was found that in addition the patient's mother had a paracentric inversion of the long arm of one chromosome 13. This appears to be the first instance in which a parent of a child with a 13q- deletion has been found to have a chromosome abnormality affecting a chromosome 13. This inversion of the chromosome in the mother appears to have resulted in the partial deletion of the long arm chromosome 13 in her daughter. (2 figures, 5 references)—David Shoch

MELATONIN SYNTHESIS BY THE RETINA. Gern, W. A., and Ralph, C. L. (Dept. Zool. and Entomol., Colorado State Univ., Fort Collins, Colo.). Science 204:183, 1979.

Melatonin is a pineal secretion with a property of lightening melanocytes apparently by acting as an antagonist for the pituitary melanocyte stimulating hormone. It may thus reasonably be considered a hormone and until recently has been thought to be secreted only by the pineal. The authors incubated trout retinas in a whole-organ culture system and showed that these retinas in vitro synthesized tritiated melatonin from a substrate containing tritiated serotonin. This not only further interrelates the pineal and the visual organs but raises the possibility that at least in the trout the retina is an endocrine organ. (2 figures, 32 references)—David Shoch

Toxicology and Applied Pharmacology

OCULAR TOXICITY STUDIES WITH ZINC PYRIDINETHIONE. Cloyd, G. G., Wyman, M., Shadduck, J. A., Winrow, M. J., and Johnson, G. R. (Procter & Gamble Co., Miami Valley Lab., Cincinnati, Ohio). Toxicol. Appl. Pharmacol. 45:771, 1978.

Zinc pyridinethione is a fungicide and bactericide and is used in shampoos for its anti-dandruff activity. It was administered orally to a random source of domestic cats and standard beagle dogs both of which have a tapetum lucidum which is cellular in nature and the drug was also administered to atapetal beagle dogs and rhesus monkeys which do not have such a modified choroid. The zinc pyridinethione in the dose administered resulted in ocular lesions in the cat and in the standard beagle dog. The initial lesions consisted of a degeneration of tapetal cells, soon accompanied by tapetal inflammation which progresses to retinitis, intraretinal hemorrhage, and subretinal edema, resulting in retinal detachment and blindness. No gross or histopathologic ocular lesions occurred in the atapetal beagle dogs or rhesus monkeys. Apparently the tapetum lucidum is the target tissue for the ocular toxicity of zinc pyridinethione. (12 figures, 1 table, 10 references)—David Shoch

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

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For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least three months before the date of occurrence.

X CONGRESS OF YUGOSLAV OPHTHALMOLOGISTS: PREVENTION OF DISEASES AND INJURIES OF THE VISION ORGAN

The X Congress of Yugoslav ophthalmologists will be held in Ohrid, Macedonia, Yugoslavia, Oct. 6-9, 1979. The topic of the Congress is Prevention of diseases and injuries of the vision organ. For further information, write Prim Dr. Sc. Dimitar Kotevski, M.D., Medical Center—Bitola, Department of Ophthalmology, 97000 Bitola, Yugoslavia.

UNIVERSITY OF MICHIGAN MEDICAL CENTER: OCULO-PLASTIC SURGERY CONFERENCE

The Towsley Center, University of Michigan Medical School, Ann Arbor, Michigan, will sponsor an Oculo-Plastic Surgery Conference, July 19 and 20, 1979. The fee is $120 for practicing physicians; $65 for physicians-in-training. For further information write, Floyd C. Pennington, Director, Office on Continuing Medical Education, The Towsley Center, University of Michigan Medical School, Ann Arbor, MI 48019.

COLOMBIAN SOCIETY OF OPHTHALMOLOGY: XIX CONGRESS

The Colombian Society of Ophthalmology will hold the XIX Congress, Aug. 11-15, 1979, in Bucaramanga, Colombia. The subject will be Prevention and Management of Surgical Complications in Ophthalmology. For further information write, 19th Colombian Congress of Ophthalmology, Carrera 33 No. 49-35 Of. 109, Bucaramanga, Colombia.

EDWARD S. HARKNESS EYE INSTITUTE: A BASIC SCIENCE COURSE FOR RESIDENTS OF OPHTHALMOLOGY

The Edward S. Harkness Eye Institute, Columbia-Presbyterian Medical Center, will hold a Basic Science Course for residents of ophthalmology Sept. 4 to Dec. 21, 1979, from 8 A.M. to 12:30 P.M. daily. The fee is $600. For further information, write Miss M. Bowen, 635 W. 165th St., New York, NY 10032; telephone (212) 694-2725.

UNIVERSITY OF ILLINOIS: CONTINUING EDUCATION COURSE

The University of Illinois, Chicago, will offer a continuing education course, Standards of Eye Care for the Primary Physician: Office and Emergency Ocular Care, will be held at the University of Illinois, Chicago, Sept. 13 and 14, 1979. For further information, write Ms. Debbie Huml, University of Illinois at the Medical Center, Office of Continuing Medical Education Services, 1853 W. Polk St., Room 144, Chicago, IL 60612.

EDWARD S. HARKNESS EYE INSTITUTE TENTH ANNUAL COURSE: THE MANAGEMENT OF APHAKIA

The Tenth Annual Course of the Edward S. Harkness Eye Institute will be held Sept. 27 and 28, 1979. Emphasis will be on the management of infantile aphakia, the complications of aphakia and the use of spectacles, contact lenses, extended-wear contact lenses and intraocular lenses. For further information, write Elizabeth Gerst, Columbia University, College of Physicians and Surgeons Continuing Education Center, 630 W. 168th St., New York, NY 10032.

UNIVERSITY OF TEXAS HEALTH SCIENCE CENTER: OPTICS, REFRACTION, AND CONTACT LENSES

The University of Texas Health Science Center at San Antonio will present a Continuing Medical Education course, "Optics, Refraction, and Contact Lenses," Sept. 14 and 15, 1979. For further information, write Office of Continuing Education Services, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78284; telephone (512) 691-7291.

TULANE MEDICAL CENTER: ANTERIOR AND POSTERIOR SEGMENT VITRECTOMY

A course for Anterior and Posterior Segment Vitrectomy will be held Sept. 14 and 15, 1979, at the Tulane Medical Center, New Orleans, Louisiana. The fee is $295, which includes all lectures, animal surgery workshops, materials, lunches and dinner in the French Quarter for the registrant and a guest.

For further information, write the Office of Continuing Education, Tulane Medical Center, 1430 Tulane Ave., New Orleans, LA 70112.

SOUTHWEST FOUNDATION FORUM AND THE NATIONAL EYE INSTITUTE: SYMPOSIA ON THE USE OF NONHUMAN PRIMATES IN VISION RESEARCH

The second in a series of symposia on the Use of Nonhuman Primates will be held Jan. 27-30, 1980, in San Antonio, Texas. This symposium is sponsored by the Southwest Foundation Forum and the National Eye Institute. Attendance is limited to 150 investigators. The registration fee is $50. For further information, write S. S. Kalter, Ph.D., Organizing Secretary, Department of Microbiology and Infectious Diseases, Southwest Foundation for Research and Education, P.O. Box 28147, San Antonio, TX 78284.

CONRAD BERENS LUNCHEON AND CONRAD BERENS MEDAL AND CERTIFICATE

At the national meeting of the Contact Lens Association of Ophthalmologists, Inc. in January 1979, Jorge N. Buxton was awarded the Conrad Berens Medal and Certificate. Dr. Buxton was chosen to deliver the Eleventh Conrad Berens Memorial Lecture "Postsurgical Visual Rehabilitation."

The annual Conrad Berens lectureship honors the recipient for his outstanding achievements in corneal and contact lens work. Dr. Buxton is Director of the Cornea Clinic of the New York Eye and Ear Infirmary.

ALAN M. LATIES AND RALPH W. RYAN APPOINTED TO THE NATIONAL ADVISORY EYE COUNCIL

Donald S. Fredrickson, Director of the National Institutes of Health announced the appointments of Alan M. Laties, and Ralph W. Ryan to the National Advisory Eye Council.

As members of the Council, Drs. Laties and Ryan will advise the National Eye Institute Director and staff on the awarding of grants for research and research training related to the eye and visual disorders, and also help provide guidance on program policy, planning, and development.

Dr. Laties is both Director of Research of the Scheie Eye Institute and the Irene Heinz Given and John LaPorte Given Professor of Ophthalmology at the University of Pennsylvania School of Medicine. Dr. Ryan, Director of the Morgantown Eye Clinic in West Virginia played a key role in the creation of the National Eye Institute.

PERSONAL

A. EDWARD MAUMENEE

A. Edward Maumenee has been awarded the Krieger Prize on Ophthalmology from Sinai Hospital of Baltimore. Dr. Maumenee was cited for his outstanding contributions in the field of ophthalmology.

NEW RESEARCH GRANTS AWARDED BY THE NATIONAL EYE INSTITUTE

January 1, 1979, to March 31, 1979

RESEARCH PROJECT GRANTS

INSTITUTION	PRINCIPAL INVESTIGATOR	TITLE OF PROJECT	COSTS AWARDED
University of California San Diego, California	Ruben Adler	Visual neurons: development and regeneration in vitro	$ 80,683
SRI International Menlo Park, California	William S. Baron	Retinal information processing	85,755
University of Wisconsin Madison, Wisconsin	George H. Bresnick	Electroretinographic studies in diabetic retinopathy	79,711
University of Iowa Iowa City, Iowa	Thomas C. Burton	Macular photocoagulation study	49,985
Louisiana State University Medical Center New Orleans, Louisiana	Ysolina M. Centifanto	HSV strain specificity and ocular disease	58,183

INSTITUTION	PRINCIPAL INVESTIGATOR	TITLE OF PROJECT	COSTS AWARDED
University of California Berkeley, California	Theodore E. Cohn	Psychophysical analysis of visual sensory deficit	$ 27,459
Yale University New Haven, Connecticut	Edward M. Cotlier	Insulin & growth hormone receptors in retinal vessels	100,645
Boston Biomedical Research Institute Boston, Massachusetts	Peter F. Davison	The collagens in lamellar tissues of the eye	131,227
Hebrew University of Jerusalem Jerusalem, Israel	Shabtay Dikstein	Gaba and the rabbit corneal endothelial pump	29,700
University of Illinois Medical Center Chicago, Illinois	Clyde C. Doughty	Cataract enzyme aldose reductase active site	55,815
Medical College of Wisconsin Milwaukee, Wisconsin	Arnold I. Goldman	Factors controlling disc shedding and phagocytosis	65.202
Yale University New Haven, Connecticut	Dale S. Gregerson	The nature and role of antiimmune responses in uveitis	75,434
Virginia Commonwealth University Richmond, Virginia	Thomas M. Harris	Ocular basement membrane changes in corneal pathology	59,042
Wayne State University Detroit, Michigan	Linda D. Hazlett	Alteration with age of resistance to eye infections	51,416
Eye and Ear Hospital of Pittsburgh Pittsburgh, Pennsylvania	Winston W. Kao	Biosynthesis of collagen in embryonic chick cornea	54,287
Albany Medical College Albany, New York	Pei-Fei-Lee	Studies of aqueous humor of glaucomatous eyes	74,511
University of California San Francisco, California	Harold F. Leeper	Intracellular recording from mammalian photoreceptors	38,620
Boston University Boston, Massachusetts	Howard M. Leibowitz	Development of functional biopolymers for the eye	92,961
University of California Irvine, California	Irving H. Leopold	Characterization of tear protease inhibitors	44,377
University of Massachusetts Amherst, Massachusetts	A. Bruce Mac Donald	Ocular immunology and pathogenesis of trachoma	85,056
Johns Hopkins Hospital Baltimore, Maryland	Robert W. Massof	Studies of visual function in the peripheral retina	46,025
Case Western Reserve University Cleveland, Ohio	Gheorghe D. Mateescu	NMR spectral studies of visual pigments	87,624
University of Sydney Sydney, Australia	Johnston W. Mc Avoy	Studies on lens cell differentiation	41,513

INSTITUTION	PRINCIPAL INVESTIGATOR	TITLE OF PROJECT	COSTS AWARDED
Eye and Ear Hospital of Pittsburgh Pittsburgh, Pennsylvania	Bartly J. Mondino	Lymphocytes in immunologic diseases of the eye	$ 87,578
University of Wisconsin Madison, Wisconsin	Vicente M. Montero	Normal and abnormal visual pathways in mammals	83,183
Eye Research Institute of Retina Foundation Boston, Massachusetts	Noritsugu Mukai	Retinal tumor cells lines in two animal models	52,697
Estelle Doheny Eye Foundation Los Angeles, California	Anthony B. Nesburn	Ocular herpes simplex infection in rabbits	136,953
University of Southern California Los Angeles, California	Nicholas Nicolaides	External eye disease and meibomian gland libids	81,639
University of Texas Health Science Center Houston, Texas	Michael D. Oberdorfer	Eye development and abnormal visual pathways	24,914
University of Texas Health Science Center Dallas, Texas	John G. Parnavelas	Visual cortex development	65,973
University of California San Francisco, California	Jon R. Polansky	Trabecular meshwork cell culture	110,115
Harvard University Boston, Massachusetts	Elio Raviola	Cell communication in retina and lens	99,800
Wayne State University Detroit, Michigan	Howard Rothstein	The cell cycle of corneal endothelium	38,305
Louisiana State University Medical Center New Orleans, Louisiana	Jerry W. Smith	Viral antigens in herpetic eye disease	74,780
Johns Hopkins University Baltimore, Maryland	William S. Stark	Normal and anomalous visual receptor function	56,776
Presbyterian-University of Pennsylvania Philadelphia, Pennsylvania	Richard A. Stone	Anion transport in the iris and ciliary body	76,268
Harvard University Boston, Massachusetts	Robert B. Szamier	Morphological studies of retinal degenerations	66,096
Johns Hopkins University Baltimore, Maryland	David L. Valle	Gyrate atrophy: treatment and pathophysiology	128,194
Institutes of Medical Sciences San Francisco, California	David W. Vastine	Experimental studies of adenoviral ocular infections	123,461
City College of New York New York, New York	Joshua Wallman	Role of accessory optic system in oculomotor control	47,759

INSTITUTION	PRINCIPAL INVESTIGATOR	TITLE OF PROJECT	COSTS AWARDED
Yale University New Haven, Connecticut	Talbot H. Waterman	Photoreceptor membrane turnover controls	$ 96,417
University of Alabama Birmingham, Alabama	David R. Whikehart	Metabolic control of corneal deturgescence	59,750
University of Alabama Birmingham, Alabama	Graeme S. Wilson	Light scattering by the corneal epithelium	12,610
University of Louisville Louisville, Kentucky	Kai-Lam W. Wun	Initiation of light-induced lesions of the retina	10,000

CORE CENTER GRANTS

INSTITUTION	PRINCIPAL INVESTIGATOR	TITLE OF PROJECT	COSTS AWARDED
Louisiana State University Medical Center New Orleans, Louisiana	Herbert E. Kaufman	Pharmacology and external ocular diseases	$151,492

NATIONAL RESEARCH SERVICE AWARD FELLOWSHIPS

INSTITUTION	PRINCIPAL INVESTIGATOR	PROJECT TITLE	COSTS AWARDED
University of Florida Gainesville, Florida	Beth R. Friedland	Localization of carbonic anhydrase in ocular tissues	$ 13,000
U.S. National Institutes of Health Bethesda, Maryland	Carole H. Latker	Effects of vitamin E on ocular angiopathy	11,000
University of California San Francisco, California	David J. Schanzlin	Research fellowship in corneal immunological diseases	15,800

RESEARCH CAREER DEVELOPMENT AEARDS/ ACADEMIC INVESTIGATOR AWARDS

INSTITUTION	PRINCIPAL INVESTIGATOR	TITLE OF PROJECT	COSTS AWARDED
Harvard University Boston, Massachusetts	Susan Y. Schmidt	Biochemical studies of retinal degenerations	$ 38,621

INSTRUCTIONS TO AUTHORS

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THE AMERICAN JOURNAL OF OPHTHALMOLOGY publishes original and timely articles dealing with clinical and basic ophthalmology. Every typescript submitted is evaluated by two or more scientific referees who recommend that the paper be (1) accepted as submitted, (2) returned for revision, or (3) rejected. Acceptance is determined by such factors as the originality, significance, and validity of the contribution; the suitability of the subject matter for subscribers of THE JOURNAL; and the editorial care with which the manuscript has been prepared. Manuscripts that require extensive editorial correction or mechanical preparation will be returned for that purpose.

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Single spacing should not be used anywhere in the manuscript. The entire manuscript—including title page and footnotes, text, acknowledgments, references, tables, and legends—should be double spaced. Vertical lines and underlining should not be used anywhere in the text, references, tables, or legends. See the section on tables for the use of horizontal lines.

Submit an original and at least one duplicate copy of both the typescript and the figures. Xerographic duplicates of the typescript are preferred to carbon copies.

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2. Text and summary
3. Acknowledgments
4. References
5. Tables
6. Legends for figures

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2. Bjorklund, A., Flack, B., and Owman, C.: Fluorescence microscopic and microspectrofluorometric techniques for the cellular localization and characterization of biogenic amines. In Berson, S. A. (ed.): Methods in Investigative and Diagnostic Encocrinology, vol. 1. London, North-Holland Publishing Co., 1972, p. 318.

3. Duke-Elder, S., and Leigh, A. G.: Diseases of the Outer Eye. Cornea and Sclera. In Duke-Elder, S. (ed.): System of Ophthalmology, vol. 8, pt. 2. St. Louis, C. V. Mosby, 1965, pp. 811-856.

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A Manual of Style, 12th ed. Chicago, University of Chicago Press, 1969.

Strunk, W., Jr., and White, E. B.: The Elements of Style, 3rd ed. New York, Macmillan Co., 1979.

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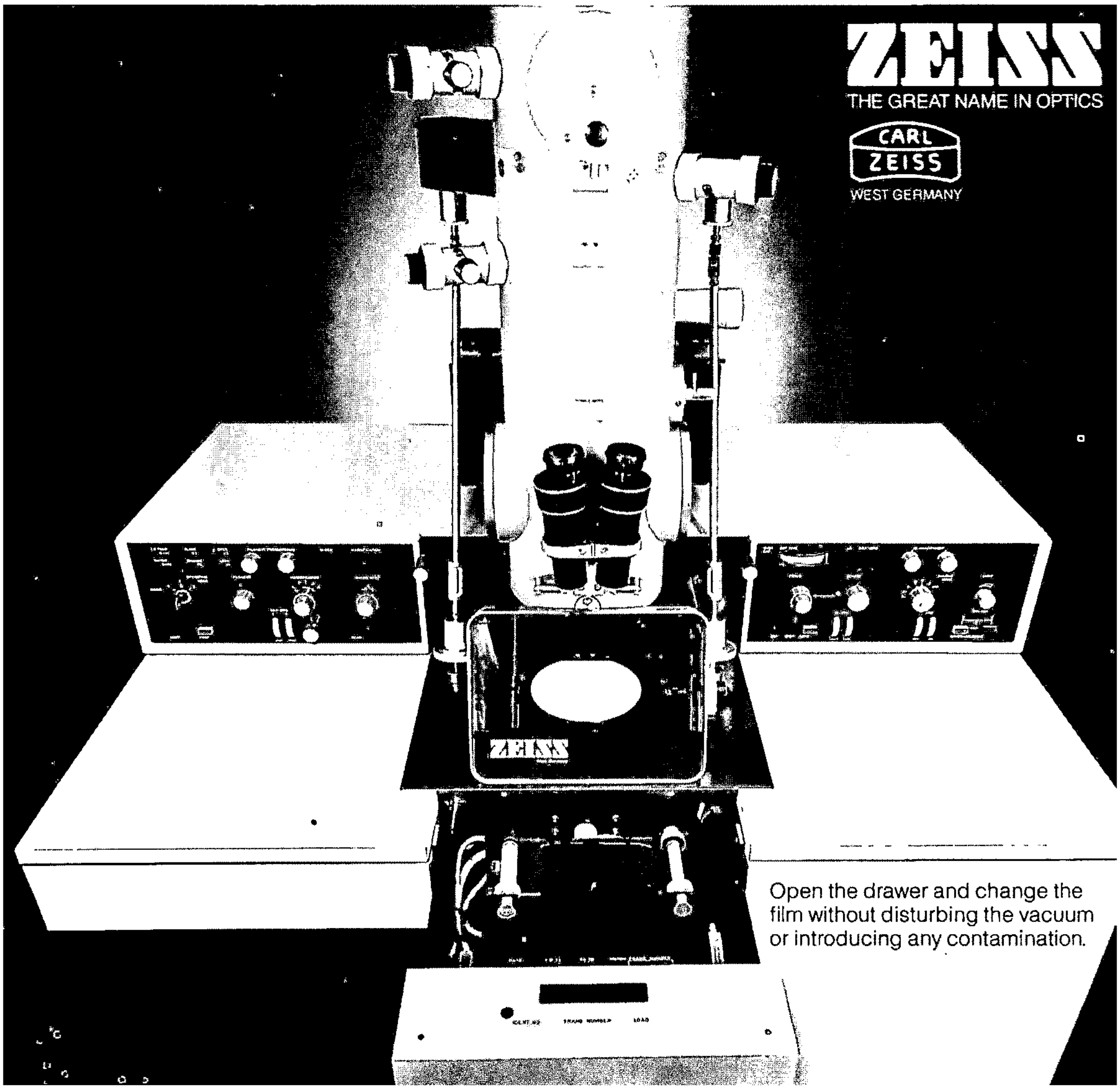

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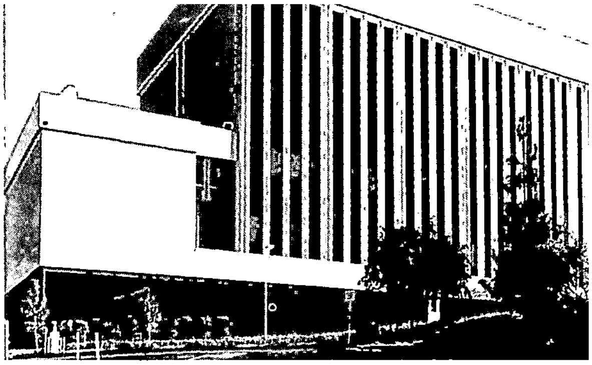

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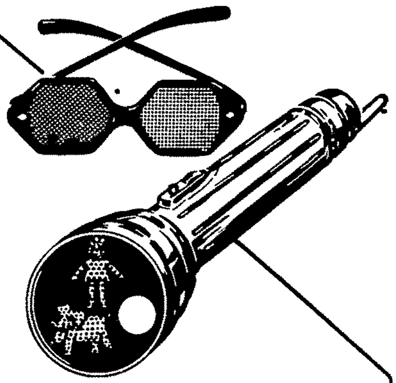

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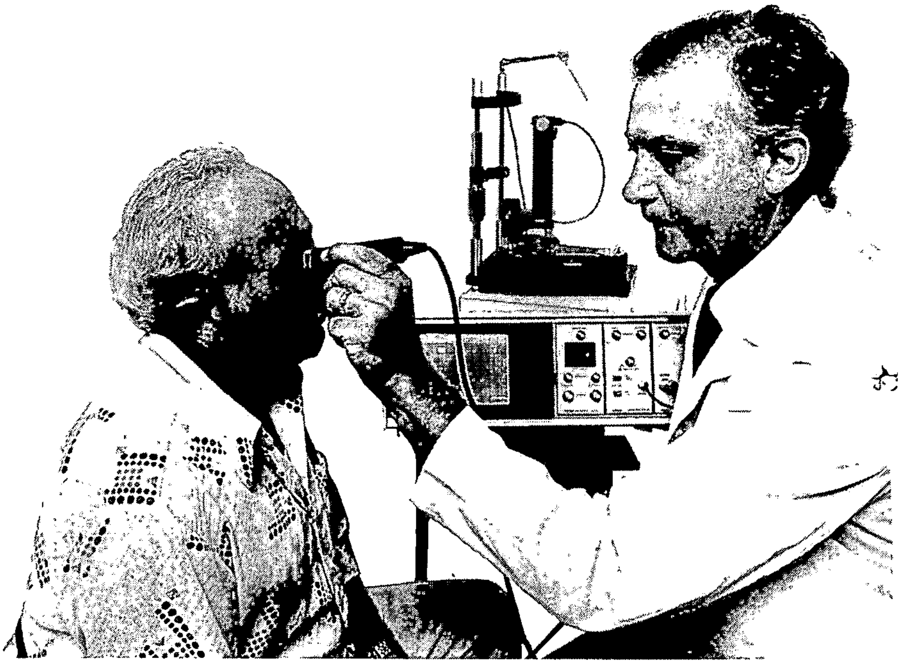

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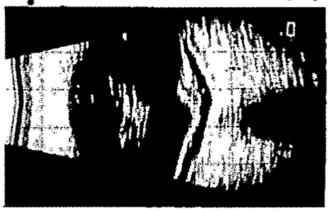

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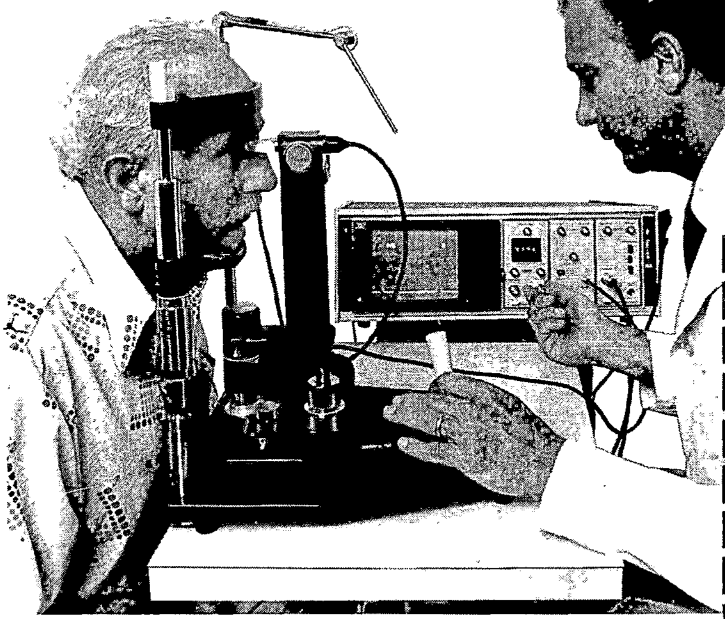

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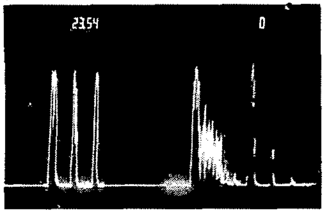

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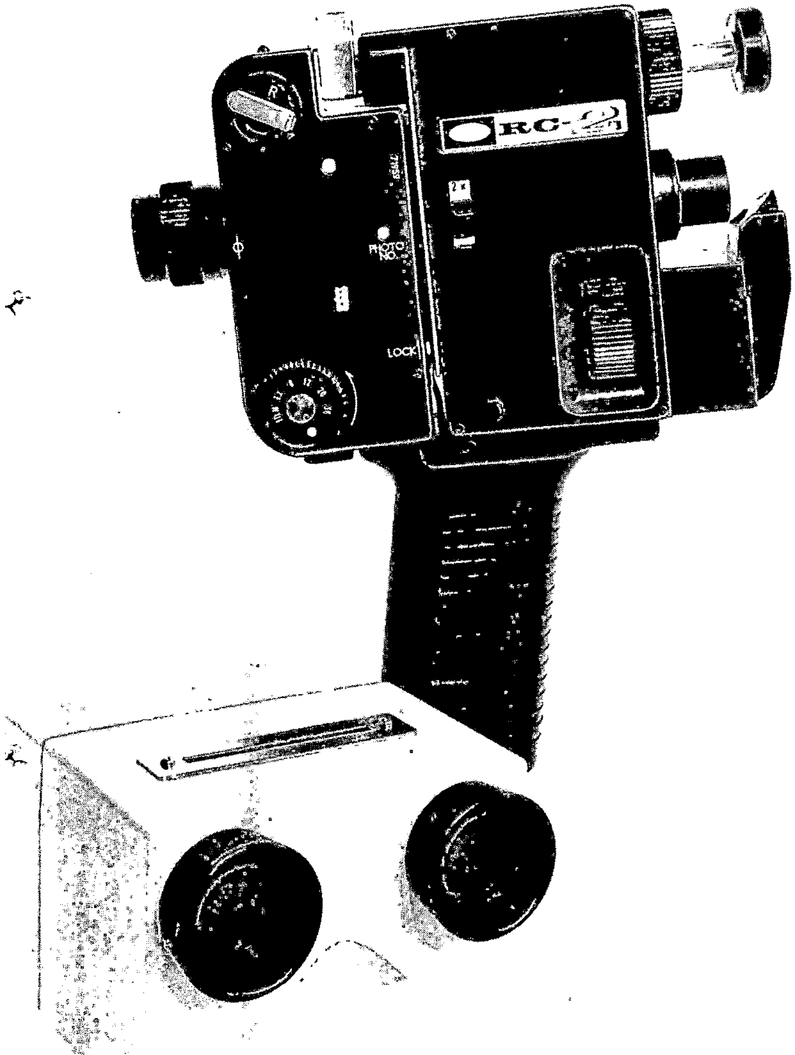

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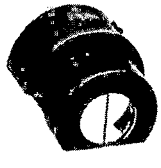

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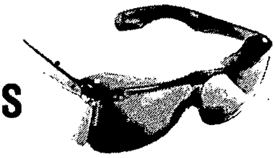

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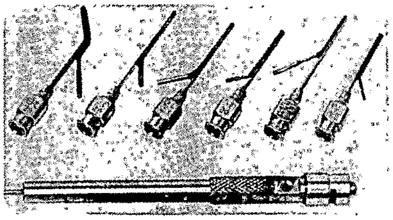

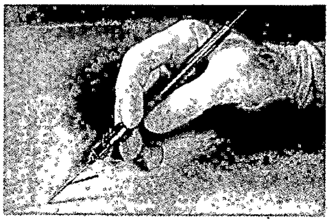

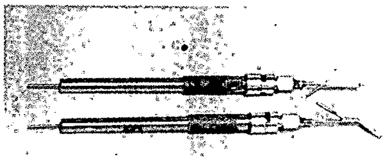

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OPHTHALMOLOGIST: Needed July 1980 to join ophthalmologist and optometrist in established and growing department of a 40-man dynamic multispecialty clinic in Madison, Wisconsin. Clinic is in a key downtown location and is expanding by adding two clinics in new high population areas adjacent to brand new regional shopping centers. Excellent opportunity for right individual. Send CV to Box 205 AJO

FOR SALE: Eastern Pennsylvania. Lucrative, established, suburban general ophthalmology practice. Large surgical volume. Short distance Baltimore, Philadelphia, Washington. Excellent opportunity for recent graduate or established practitioner seeking relocation. Box 206 AJO

VISUAL SCIENTIST/BIOMEDICAL ENGINEER: Ph.D. August 1979 desires challenging academic position. Experience includes seven years clinical electrophysiology, basic research in contrast sensitivity and Westheimer curves. Instrument development, resident teaching, publications, NIH grant. Box 207 AJO

OPHTHALMOLOGIST: To join active established medical and surgical practice in a young, multi-specialty clinic. Small college community in Appalachian Mountain foothills with outstanding family living. Attractive compensation package and working conditions. Contact: Richard A. Callis, Administrator, Morehead Clinic, 234-R Flemingsburg Road, Morehead, KY 40351

FELLOWSHIP IN OPHTHALMIC MICROSURGERY: Immediately. Bilingual (English/Spanish) preferred. $1,000 per month. Louis J. Girard, M.D., 4126 S.W. Freeway, Suite 600, Houston, TX 77027

POSITION AVAILABLE: Director of Education, Department of Ophthalmology, with active residency in community hospital. Contact Medical Director, Maryland General Hospital, 827 Linden Avenue, Baltimore, MD 21201

GENERAL OPHTHALMOLOGIST: Needed to join busy surgically oriented department in rapidly expanding multispecialty clinic of 100 physicians; adjacent hospital in large university community of 100,000; outstanding fringe benefits and salary leading to full associateship; teaching affiliation with medical school; Send CV to Medical Director, Carle Clinic Association, 602 W. University Avenue, Urbana, IL 61801

POSITION AVAILABLE: Combined Program in Ophthalmology, University of Alabama in Birmingham-Eye Foundation Hospital. Full-time faculty member with subspecialty interest in glaucoma. Responsibilities include; teaching residents and medical students, patient care, and research. Considerable opportunity for individual professional development in all phases of academic involvement. One year fellowship training required, preferably with basic or basic clinical research experience. Salary commensurate with experience of appointee. Affirmative Action, Equal Opportunity Employer. Contact: Roswell R. Pfister, M.D., Program Combined in Ophthalmology, 1720-8th Avenue, South, Birmingham, AL 35233

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FELLOWSHIP IN MEDICAL RETINA: To conduct original clinic and some laboratory research on diabetic retinopathy. Minimum requirement at least PG-3 in Ophthalmology. Position available July 1, 1979. Send resume to: Albert M. Potts, Ph.D., M.D., Professor and Chairman, Department of Ophthalmology, University of Louisville, 301 East Walnut Street, Louisville, KY 40202. The University of Louisville is an affirmative action/equal opportunity employer.

MEDICAL OFFICE-NEW JERSEY-BERGEN COUNTY: Podiatrist has 9 room office with furnished waiting room to share with ophthalmologist or optometrist. Busy shopping center, ample parking. Immediate occupancy. Reasonable rent. Larry Price, D.P.M., 636 Westwood Avenue, River Vale, New Jersey, 07675 (201) 666-5115.

PRACTICE AVAILABLE: Western Colorado. Contact Nicholas Cole, Box 1909, Montrose, CO 81401. Laser and Phaco 75-bed hospital across from office. Excellent recreational access. Good family atmosphere. Stay to introduce.

FULL-TIME FACULTY POSITION: Subspecialty in diseases and surgery of the retina and vitreous. Board-eligible or certified. Generous practice opportunities and income. Academic appointment commensurate with experience. Immediate need. An equal opportunity/affirmative action employer. Write or call: Stuart I. Brown, M.D., Professor and Chairman, Department of Ophthalmology, University of Pittsburgh School of Medicine, Eye and Ear Hospital, 230 Lothrop Street, Pittsburgh, PA 15213. (412) 647-2205

OPHTHALMOLOGIST WANTED: To take over an established general ophthalmology practice in a predominantly Spanish-speaking area of San Antonio, Texas. Ability to speak spanish helpful but not essential. No investment required. Any arrangement negotiable. Contact James W. Speights, M.D., 700 S. Zarzamora, Suite 302, San Antonio, TX 78207. Phone: (512) 432-0979.

CHAIRPERSON, DEPARTMENT OF OPHTHALMOLOGY: Applications are invited from qualified M.D.'s with demonstrated competence in ophthalmology for the position of Chairperson, Department of Ophthalmology, Indiana University School of Medicine. Interested parties should send their curriculum vitae to Arthur L. Norins, M.D., Chairman, Search and Screen Committee, Indiana University School of Medicine, 1100 West Michigan Street, Indianapolis, IN 46223. Indiana University is an affirmative action and equal opportunity employer.

WANTED: Vitreoretinal Fellow, New York City, 12 month program, July 1979, NIH Research Grants. Retinal detachment and vitreous surgery, photocoagulation, fluorescein angiography, ultrasound. Inquire: David B. Karlin, M.D., 132 East 76th Street, New York, NY 10021

FOR SALE: Da-Laur Ophthalmic exam table with side arm and pillow, black, $150; B & L Heat-treat lens hardening unit, $200; Standard drop ball test, $25; old B & L Ferrie Rand perimeter and A/Owottring troposcope and slides. David Fenske, M.D., 412 Phoenix Street, South Haven, MI 49090

CLINICAL FELLOWSHIPS: Offered by University of Illinois, Department of Ophthalmology beginning July 1979 for 1-2 years. 1. Retina/Vitreous-Diabetes—Includes fluorescein angiography, photocoagulation, retina and vitreous surgery. 2. Retinal Vascular Diseases-Sickle Cell—Includes fluorescein angiography, ultrasonography, photocoagulation. 3. Medical Retina/Inherited Diseases-Genetics—Includes fluorescein angiography, electrophysiology. 4. Glaucoma Research/Screening—Includes glaucoma research and Lions Club-sponsored glaucoma screening. Applicants should have or be able to obtain an Illinois license. Please send curriculum vitae to: Morton F. Goldberg, M.D., Professor and Head, Department of Ophthalmology, University of Illinois, Eye and Ear Infirmary, 1855 W. Taylor Street, Chicago, Illinois 60612

OPHTHALMIC TECHNICIAN: Under general supervision of ophthalmologists in a major teaching clinic with ophthalmology residency, and NIH funded clinical research. Responsibilities will include: performs documenting and measurement tasks as requested; assists in performance of complex clinical procedures; and tasks in orthoptics as necessary. Must have successfully completed an ophthalmic technician program or completed a program to become a certified orthoptist. Preference given to experience in field. Please send resume to: Employee Relations Department, Good Samaritan Hospital and Medical Center, 1015 N.W. 22nd Avenue, Portland, Oregon 97210. An equal opportunity employer.

VITREO-RETINAL SURGEON: Two year VR fellowship, three years academic and clinical experience, medical and surgical retina, diabetes, ultrasound. Northeast, midwest southeast. Box 208 AJO

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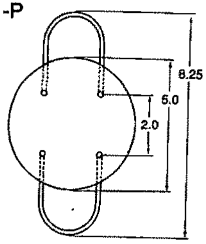

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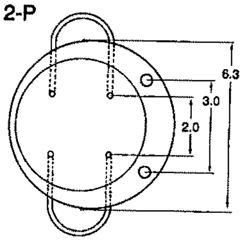

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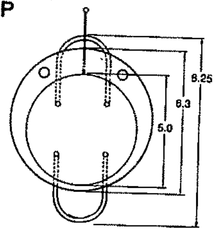

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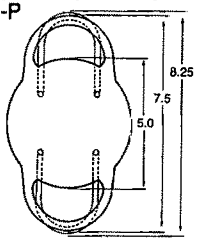

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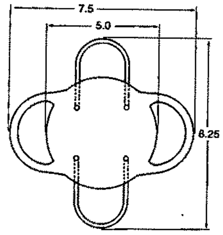

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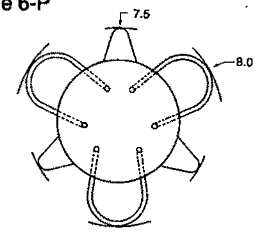

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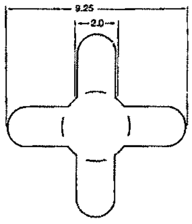

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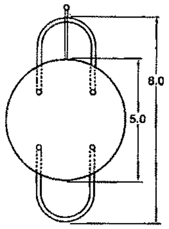

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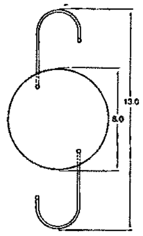

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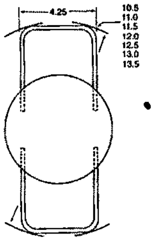

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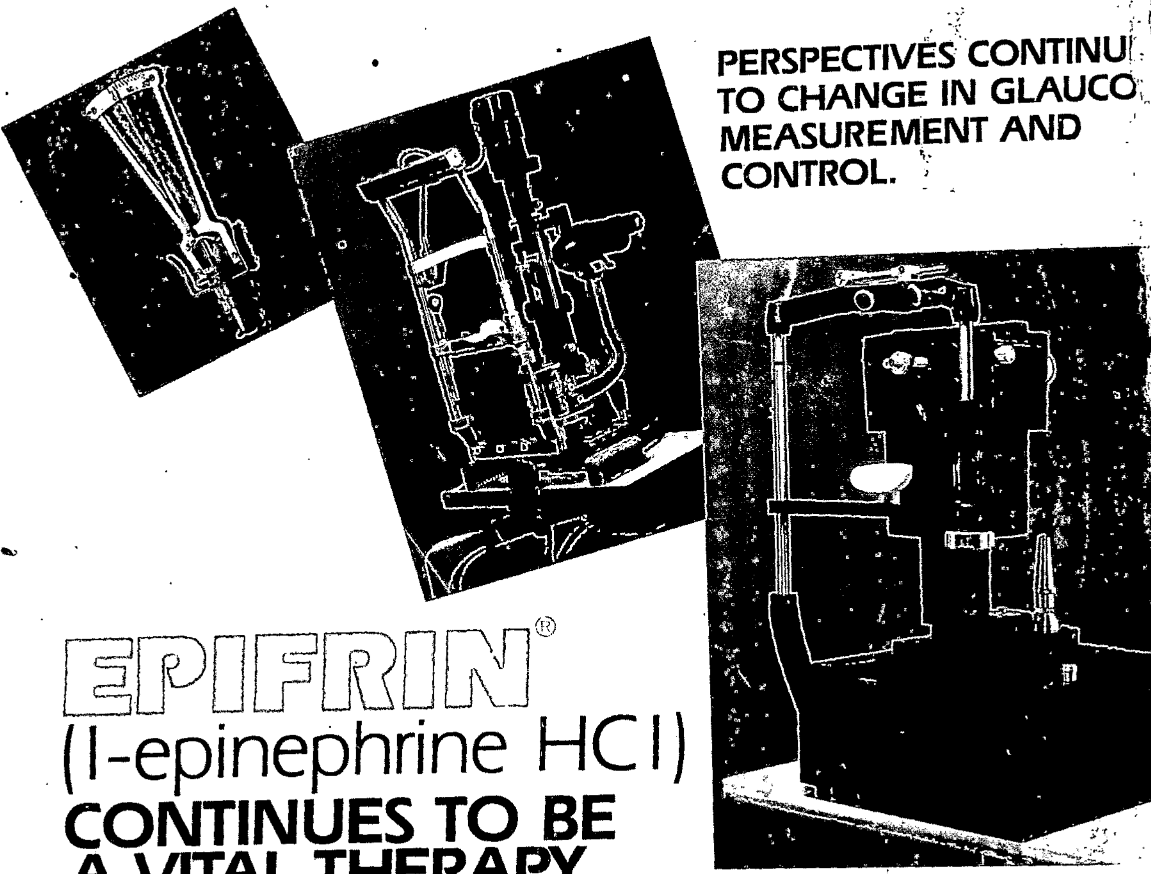

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1. Obstbaum S.A. et al, Timolol: Effect on intraocular pressure in chronic open-angle glaucoma. Ann Ophthalmol 10(10): 1347-1351, 1978.
2. Katz, I.M., Beta-blockers and the eye: An overview. Ann Ophthalmol 10(7): 847-850, 1978.
3. A study conducted by an independent research organization. Allergan Pharmaceuticals Report Series No. 148, 1979.

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